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Chapter 9: Flaps and Skin Grafts

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INTRODUCTION

CHAPTER OBJECTIVES

After studying this chapter, the learner will be able to:

1. **Describe a pedicled and a free flap.**
2. **Assess and determine an appropriate classification for a specific flap.**
3. **Distinguish the vascular anatomy of the skin, muscle, fascia, and perforator flaps.**
4. **Integrate the concepts of angiosomes and venosomes and their effects on flap design.**
5. **Discuss flap physiology, including delay phenomenon and tissue expansion.**
6. **Assess and monitor a flap for tissue viability.**
7. **Recognize common flap complications.**
8. **Define the types of skin grafts.**
9. **Recognize skin anatomy relevant to flaps.**
10. **Integrate skin graft healing physiology into a plan of care.**
11. **Recognize signs of graft failure.**
12. **Explain relevant elements of donor site selection.**

Primary closure of a surgical wound is the simplest and fastest way of approximating wound margins; however, in many instances it is neither feasible nor desirable to close a wound with this method. Using principles of moist wound healing results in a wound being left open to heal by secondary intention. Primary closure results in minimal scarring and re-epithelialization; wounds left to heal by secondary intention may have more extensive scarring with subsequent contraction and deformity.

When there is a relative or absolute soft tissue deficit appropriate for coverage, a flap or skin graft can be used to fill the defect. The extent of the deficit and its location, among other factors, dictate the type of coverage needed. The first section of this chapter discusses and illustrates different types of flaps, followed by a succinct description of flap monitoring and common complications. The second section demonstrates the principles and applications of skin grafting.

While this chapter is not an exhaustive reference on the subject, it is intended to be comprehensive to wound care providers, focusing on essential concepts related to flaps and grafts that will help the clinician understand the indications and postoperative care. For those readers who require more detailed information on the topics presented in this chapter, a thorough reference list is provided for further study.

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FLAPS

Definition

A flap is a unit of vascularized tissue that may be transferred from one part of the body to another.[1] It is more simply defined as specific tissue that is mobilized on the basis of its vascular anatomy.[2] A flap may contain a single tissue (for example, skin, muscle, fascia, fat, bone, tendon, nerve) or a combination of tissues. A flap may also be comprised of enteric components such as jejunum, colon, stomach, or omentum. The critical concept to understand about flaps is the relationship to its blood supply, which is necessary for the flap to survive. Because of the thickness and/or composite nature of flaps, the tissue being transferred cannot initially survive by diffusion from the recipient bed; it must have its own inflow and outflow of blood. A flap is termed *pedicled* if it maintains its blood vessel continuity at all times and is raised and/or transposed on the pedicle; it is termed a *free* flap if the vein and artery of the pedicle are cut and re-anastomosed to other blood vessels. The term *free tissue transfer* may also be used to describe a *free flap*.

Classification

The dichotomy between a pedicle and a free flap is easy to understand but fails to classify flaps in a useful way. Over the years, different classifications have been used to categorize flaps according to various predominant factors. Some of the useful classifications are vascular supply, tissue composition, transfer method, and vascular orientation.

Vascular Supply

Classification by vascular supply is based on the type of vascularization of the flap and is utilized for cutaneous flaps, whether alone or combined with another tissue.

Random Flap

This concept is only applied to skin. Random cutaneous flaps are based on random nondominant contributions from the dermal and subdermal plexus.[1] Rather than having a definite blood vessel system to supply its surface, a random flap relies on the interconnection between specific vascular territories and the vascular skin plexus.[1] The critical concept in a random flap is its width-to-length ratio. A length twice as long as its base (ratio 2:1) is generally considered the limit for most tissue; however, it may be greater in the face and scalp but smaller in the lower extremity.[3] Exceeding a 2:1 length-to-width (base) ratio predictably results in distal flap necrosis. A classic example of a random flap is a transposition flap as illustrated in **FIGURE 9-1**.

FIGURE 9-1

Random flap Illustration of a random pattern flap in a transposition rectangular flap (2:1 ratio). (Used with permission of Losee JE, Gimbel M, Rubin J, Wallace CG, Wei F. Plastic and reconstructive surgery. In: Brunicardi F, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE, eds. *Schwartz's Principles of Surgery*. 10th ed. New York, NY: McGraw-Hill; 2015:chap. 45.)

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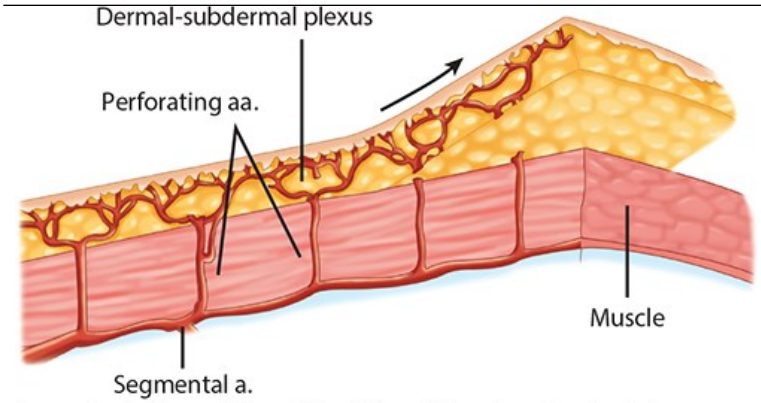

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Axial Flap

An axial flap is based on longitudinal vessels along the pathway of the flap. The significant advantage of an axial flap over a random flap is the capability to extend the design outside the 2:1 ratio. In fact, an axial flap can be as long as the length of the blood vessel included in the flap. In this type of flap, the main axial artery with its *venae comitantes* gives rise to multiple branches that feed and drain the cutaneous portion adjacent to the flap. A classic example is the groin flap, based on the superficial circumflex iliac artery.

Musculocutaneous

A musculocutaneous flap involves a combination of skin and muscle. The vascular pedicle supplies a muscle, which at the same time provides perforator branches to the skin. An example of a musculocutaneous flap is the transverse rectus abdominis flap (TRAM) used frequently in breast reconstruction.

Fasciocutaneous

In a fasciocutaneous flap, the fascia is the recipient of a named vessel. Once the artery reaches the fascia, it gives perforator branches to the skin. An example of a fasciocutaneous flap is the anterolateral thigh flap (ALT) with the lateral circumflex femoral artery (LCFA) as a pedicle. The artery reaches the vastus lateralis and its fascia before giving perforator branches to the skin.

Septocutaneous

When perforator branches do not have to go through a muscle or a fascial layer, they travel between structures. The space where these vessels travel is termed a *septum*; therefore, the term septocutaneous flap. Interestingly, the ALT flap, with the same pedicle (LCFA), may have perforator branches traveling in the septum between the vastus lateralis and the rectus femoris muscles, reaching the skin without crossing through the vastus lateralis or its fascia. In this case, the flap is labeled a septocutaneous flap.

Tissue Composition

The tissue composition classification is based on the actual tissue type involved in the flap, which can be a single tissue or a composite of tissues. The vascular supply is indirectly implied as being included in the flap and, therefore, is not named.

Single Tissue

A single tissue flap is referred to by its main tissue. For example, a flap containing only the rectus abdominis muscle is a muscle flap; a flap composed of the temporoparietal fascia is a fascial flap. A flap can also contain skin, bone, omentum, or colon. (This list is not exhaustive.)

Composition of Tissues

When more than one tissue are combined into a flap, it is labeled using the main tissues involved. Examples include a musculocutaneous flap or an

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osteofasciocutaneous flap. Many potential combinations are possible.

Transfer Method

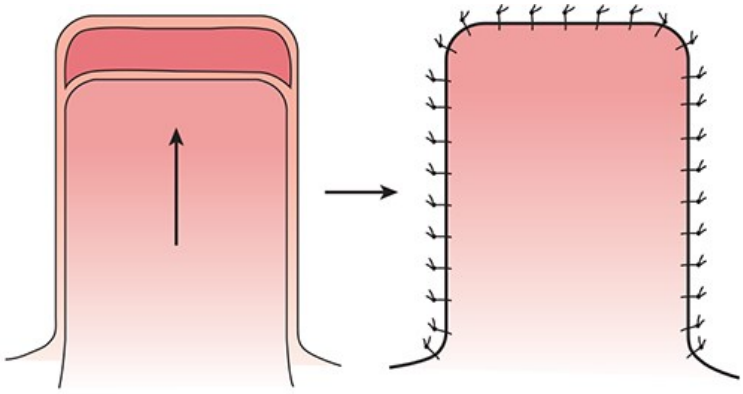
Classification by transfer method refers to the spatial displacement of a flap from a fixed point or a base. Consequently, it applies only to local pedicle flaps.

Advancement Flap

Advancement flap describes the procedure when a flap is advanced directly forward into a defect without any rotation or lateral movement (**FIGURE 9-2**).[4]

FIGURE 9-2

Example of advancement flap Advancement flap describes the procedure when a flap is advanced directly forward into a defect without any rotation or lateral movement.[4]



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Rotational Flap

A rotational flap describes the procedure in which a semicircular flap is rotated about a pivot point into the defect to be closed (**FIGURE 9-3**).[5]

FIGURE 9-3

Example of rotational flap A rotational flap is a semicircular flap that is rotated about a pivot point into the defect to be closed.[5]

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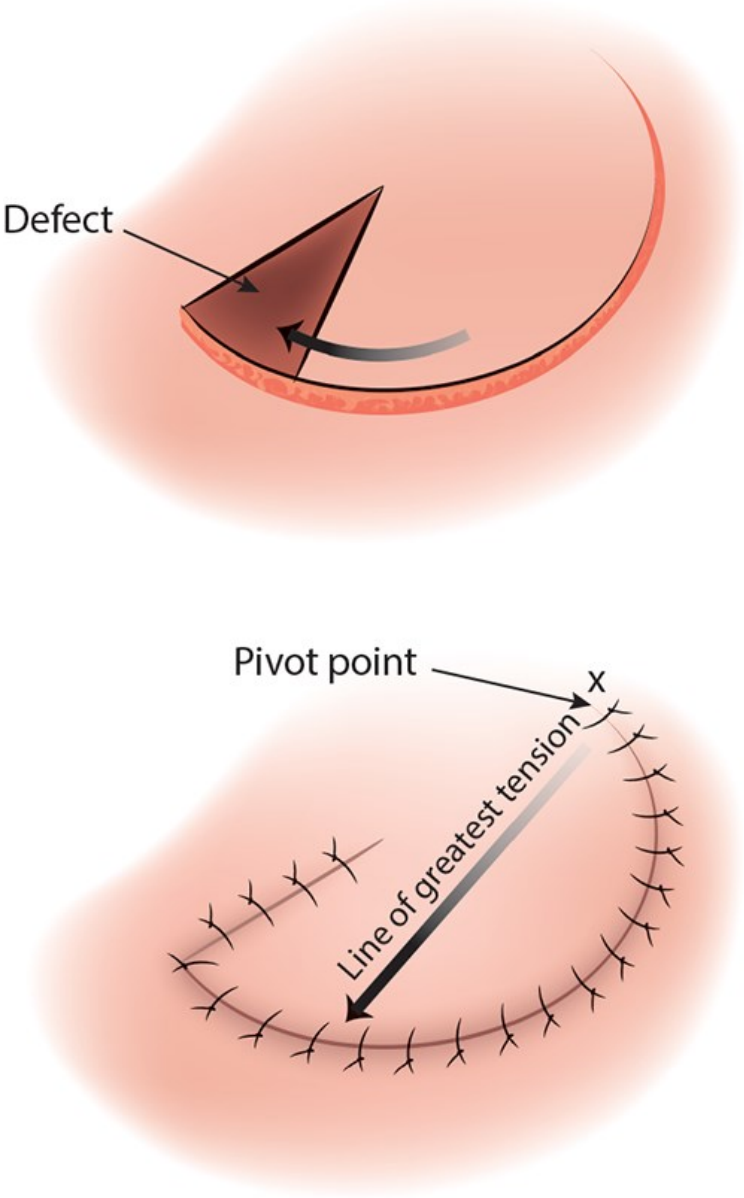

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Transposition Flap

A transposition flap is a combination of rotation and advancement of a single flap to close a defect (**FIGURE 9-4**).

FIGURE 9-4

Example of transposition flap A transposition flap is a combination of rotation and advancement of a single flap to close a defect.

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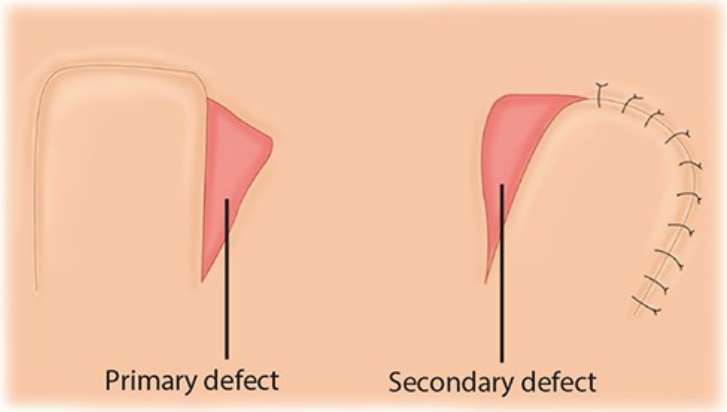

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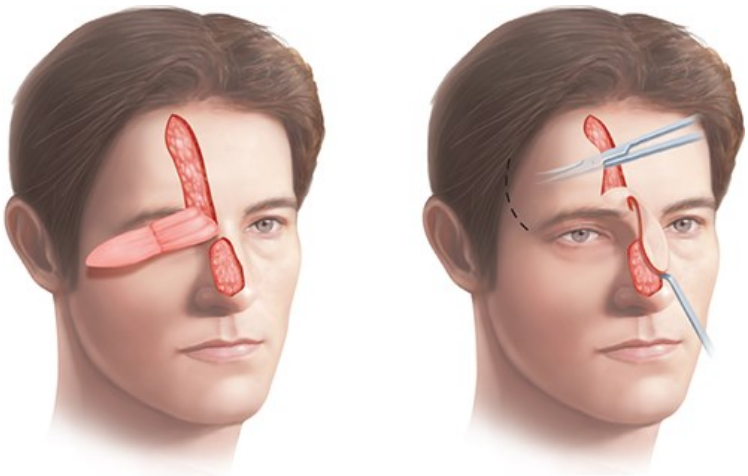
Interpolation Flap

An interpolation flap is a two-stage tissue flap in which the base of the flap is not immediately adjacent to the recipient site.[6] For this type of flap, normal tissue is interposed between the base of the flap and its insertion. The forehead flap is a classic example (**FIGURE 9-5**).

FIGURE 9-5

Example of interpolation flap An interpolation flap is a two-stage tissue flap in which the base of the flap is not immediately adjacent to the recipient site.[6] In this type of flap, normal tissue is interposed between the base of the flap and its insertion. The forehead flap is a classic example, as shown here as a paramedian forehead flap.



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Vascular Orientation

A flap can be named according to the direction of its blood flow. This classification is not specific but is used to describe a specific pattern of vascularization.

Anterograde Flow Flap

An anterograde flow flap maintains the normal pattern of the arterial flow. For example, a pedicle radial forearm flap based proximally has an anterograde arterial flow in the radial artery.

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Retrograde Flow Flap

In a retrograde flow flap, the arterial blood flow does not travel through the usual pattern but instead has its flow reversed due to transection of the anterograde dominant blood flow. An example is a reverse posterior interosseus flap in which the arterial flow in the posterior interosseus artery (PIA) originates normally from the ulnar artery and the common interosseus artery. In this flap, when the PIA is transected proximally, blood is flowing reversely from an anastomotic branch of the anterior interosseus artery located in the distal forearm.

Flow-Through Flap

A flow-through flap can be used to bridge an interrupted arterial flow by anastomosing the proximal and distal pedicle, thereby restoring flow distally. The first flow-through flap was used to connect the external carotid to the facial artery with a radial forearm flap. In this case, the radial artery was used to reestablish arterial circulation.

Venous Flap

A venous flap has no arterial pedicle, only a venous pedicle(s). The physiology of these flaps is more complex and only small flaps can survive on venous inflow only.[7]

Vascular Anatomy

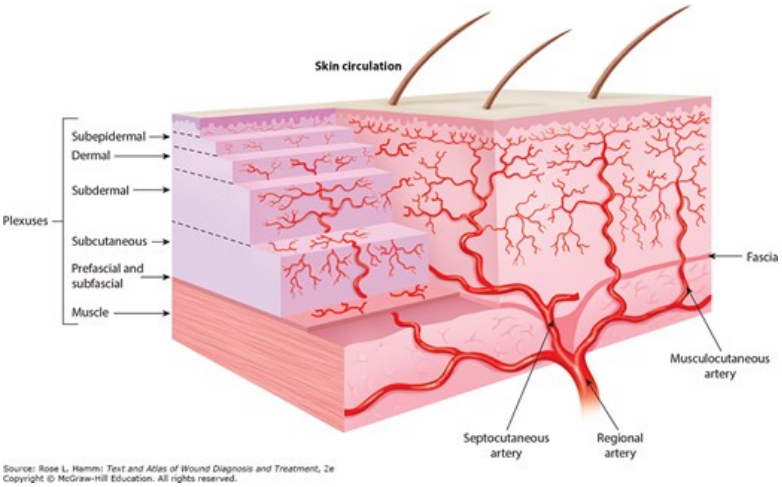
Flaps are based on an absolute understanding of their vascular supply. Without a thorough appreciation of the delicate and complex vascular anatomy of each specific tissue, the surgeon cannot harvest a flap in a safe and efficient way. Most flaps are composite flaps with each component having its own vascular territory. For the sake of clarity, in this text a *perforator artery* is defined as an artery that supplies the skin; a *branch artery* is defined as an artery that supplies a muscle or fascia.

Vascular Supply of the Skin

The skin can be nourished via direct connections from an artery; otherwise, the artery has to penetrate through various tissues, the most important being muscle and fascia. Once the artery reaches the skin, its vascular supply forms five different plexuses.[2,5] A vascular plexus can be simply described as a network of anastomoses between blood vessels (**FIGURE 9-6**).

FIGURE 9-6

Skin circulation Once the artery reaches the skin, it forms multiple different plexuses through which blood is supplied to the layers of the skin.



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The skin microcirculation can be described using another model. The arterioles and venules within the dermis form two horizontal plexuses: an upper horizontal network in the papillary dermis and a lower horizontal plexus at the dermis-subcutaneous tissue junction. Muscle and fat tissues host the perforating vessels, which ultimately create the lower plexus. The newly formed arterioles and venules connect to the upper horizontal plexus,

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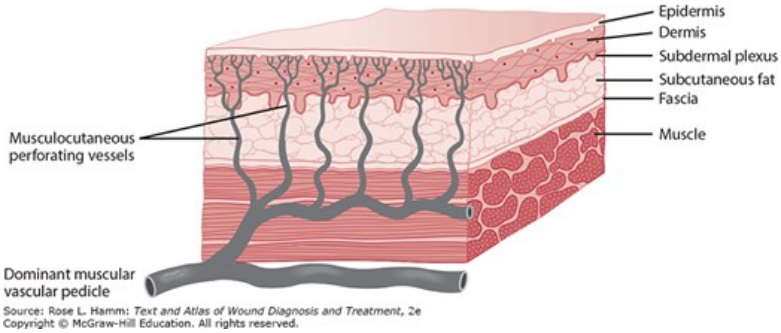
irrigating epidermal appendages at the same time (**FIGURE 9-7**).[2,6]

FIGURE 9-7

Perforator arteries to the skin The artery in the muscle provides smaller branches that perforate the fascia to provide the blood supply to the skin layers.



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Muscle Flaps

Muscle flaps are, in their simplest form, individual muscles harvested with the vascular pedicle. However, muscles are not vascularized uniformly and the understanding of the vascularization pattern is of paramount importance when harvesting a muscle flap. In a landmark article published in 1981, Mathes and Nahai described five patterns of muscle vascular anatomy (**FIGURE 9-8** and **TABLE 9-1**).[8]

Type I: One vascular pedicle. In this type of flap, the entire muscle is dependent on its main blood supply, meaning that the entire muscle can survive on a single artery and vein. The disadvantage of a type I muscle flap is that if the vascular pedicle is severed, the muscle will not survive. The *tensor fascia lata* or the *gastrocnemius* are muscles that can be used for type I flaps.[9]

Type II: One or more vascular pedicle(s) and minor pedicle(s). Muscles in this group have a dominant pedicle that can sustain the whole muscle and a minor blood supply that is incapable of solely maintaining muscle viability. In this case, a muscle flap can be harvested on a dominant pedicle and minor pedicles can be ligated without affecting the subsistence of the flap. Examples include the *gracilis* or *rectus femoris* muscles.

Type III: Two dominant pedicles. These muscles have two dominant pedicles that can each maintain adequate blood flow if harvested individually. As long as one of the two pedicles is intact, the flap will remain viable. A classic example is the *rectus abdominis* in the TRAM flap with two main vascular trunks. A pedicled TRAM uses the superior epigastric artery and veins while a free TRAM utilizes the inferior epigastric system. Another example is the *pectoralis minor* muscle, which can be harvested using the thoracoacromial artery or the lateral thoracic artery.

Type IV: Segmental vascular pedicles. These muscles are vascularized by multiple branches that are each responsible for the survival of a segment of the muscle. The origin and insertion of the muscle are considered pedicles. The primary limitation of a type IV muscle becomes obvious during flap harvest. Since each segment is not well connected to the other vascular segment, only a limited number of branches can be safely ligated and still maintain survival of the entire muscle flap. If too many branches are severed, segmental flap necrosis will occur. Examples include the *sartorius* or the *external oblique* muscles. These muscles cannot be used as a free tissue transfer but are useful as a rotational pedicle flap. Sartorius flaps, raised by ligation of two to three branches, can be used to cover an exposed femoral artery and vein.

Type V: One dominant and secondary segmental pedicles. In these muscles, the multiple segmental pedicles are capable of adequately vascularizing the flap as well as the single dominant pedicle. The flap can then be based on either of the pedicles for a pedicle flap. If free tissue transfer is performed, then the flap must be based on the dominant pedicle. Examples include the *latissimus dorsi* or the *pectoralis major*. A latissimus dorsi flap can be distally based on its lumbar branches or it can be used as a free tissue based on the thoracodorsal vascular trunk.

FIGURE 9-8

The vascular anatomy of muscles (experimental and clinical correlation) Patterns of vascular anatomy, first described by Mathes and Nahai in 1981, are also described in **Table 9-1**. (Used with permission from Mathes, N. Classification of the vascular anatomy of muscles: experimental and

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clinical correlation. *Plast Reconstr Surg*. 1981;67(2):177–187. https://journals-lww-com.ezproxy.cihe.edu.hk/plasreconsurg/Citation/1981/02000/Classification_of_the_Vascular_Anatomy_of_Muscles_.7.aspx.)

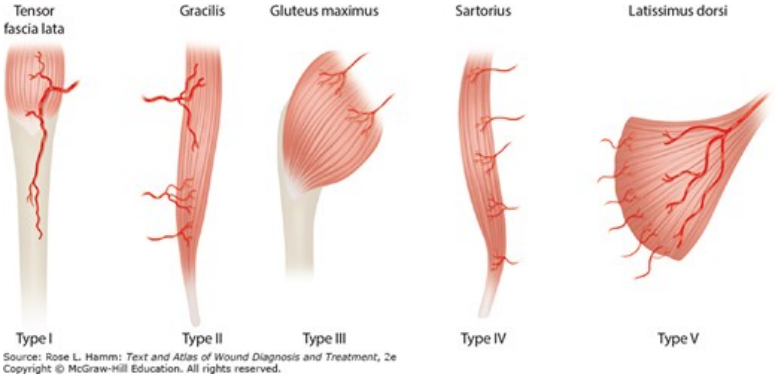

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TABLE 9-1

Patterns of Muscle Vascular Anatomy

Classification	Vascular Supply	Example
Type I	One vascular pedicle	Gastrocnemius
		Tensor fascia lata
Type II	Dominant and minor pedicles (the flap cannot survive based only on the minor pedicles)	Gracilis
		Rectus femoris
Type III	Two dominant pedicles	Rectus abdominis
		Pectoralis minor
Type IV	Segmental pedicles	Sartorius
		External obliques
Type V	One dominant pedicle with secondary segmental pedicles (the flap can survive based only on the secondary pedicles)	Pectoralis major
		Latissimus dorsi

A muscle flap can be combined with other tissues to create a composite flap, the most common being the musculocutaneous flaps. Muscles give rise to multiple arterial branches, termed perforator arteries, which travel through the fascia to supply the skin. In order to achieve better vascularization of the skin, the connections between the muscle and skin are maximized during flap surgery. Every muscle that has skin in continuity can include a portion of skin when it is used for a flap. This attached section of skin is termed a skin paddle. A rectus abdominis or a latissimus dorsi muscle can easily be combined with a skin paddle. A pectoralis minor flap, however, does not have direct connection to any skin and, therefore, cannot include a skin paddle.

Fasciocutaneous Flaps

A fasciocutaneous flap is composed of skin, subcutaneous fat, and fascia. Its main vascular supply irrigates deep fascia that permits vascularization to

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a more or less well-defined skin territory. The first fasciocutaneous flap was described by Pontén in 1981[10] and a more formal classification was developed by Cormack and Lamberty in 1984. Four types of vascular patterns were originally identified.[11]

Type A: Multiple fasciocutaneous vessels. These flaps have multiple perforators oriented in the long axis of the flap in the predominant direction of the arterial plexus and at the level of the deep fascia. An example is a rotational flap in a lower extremity.[11] As in segmental muscle flap (type IV), survival of the flap is directly related to the number of segmental vessels included in the flap.

Type B: Single fasciocutaneous vessel. These flaps have a single vessel responsible for vascularizing the entire flap. According to the authors, the essential characteristic of the flap is a T-junction on the single vessel feeding the fascial plexus.[11] An example is the medial arm flap based on the superior ulnar collateral artery.

Type C: Multiple small fasciocutaneous vessels in a septum. These flaps have branches originating from an axis vessel; however, they travel between muscles in a septum. A classic example is the radial forearm flap where the numerous perforators to the skin and fascia originate from the radial artery and travel in between the brachioradialis (BR) and the flexor carpi radialis (FCR). The main difference between type A and type C is the fact that the main vessel is included in the flap in a type C. Therefore, a type C can be converted into a free tissue transfer but a type A cannot.

Type D: Osteomyofasciocutaneous flap. This flap is an extension of type C and as such is often included in this category. In this flap, the fascial septum is included as well as the adjacent muscle and bone along with their blood supply. An example is the radial forearm osteofasciocutaneous flap including the radius bone with a cuff of muscle including fascia and skin.

Fascial flaps do not need a skin paddle and are therefore often harvested as a pure fascial flap without a skin paddle. Examples include a temporoparietal fascia flap or a radial forearm fascia flap.

For fasciocutaneous flaps, another classification by Mathes and Nahai deserves mentioning because of its simplicity and frequency of use. This classification is based on the pathway of the perforator to the skin and includes three types (**TABLE 9-2**).[2,3,5]

Type A: Direct cutaneous perforator. In these flaps, a perforator artery originates from an axial vessel and travels directly to the skin without having to travel through a muscle or a septum. Examples include the groin flap, based on perforators from the superficial circumflex femoral artery or the temporoparietal fascia based on branches from the superficial temporal artery.

Type B: Septocutaneous perforator. In these flaps, perforators travel from the originating vessel to the skin between muscles via a septum. The primary advantage of these perforators is that during harvesting of the flap, the vessel course is easily identifiable in a septum and because of the absence of side branches, dissection is rapid and straightforward.

Type C: Musculocutaneous perforators. Perforators that travel through a muscle to access the skin are termed musculocutaneous perforators. Unlike type B perforators, vessels in muscle are more difficult to dissect if the muscle is not included in the flap. Close proximity of muscle fibers to blood vessels and numerous small branches makes dissection of perforators more tedious.

TABLE 9-2

Nahai–Mathes Classification of Fasciocutaneous Flaps

Classification	Vascular Supply	Example
Type A	Direct cutaneous vessel that penetrates the fascia	Temporoparietal fascial flap
Type B	Septocutaneous vessel that penetrates the fascia	Radial artery forearm flap
Type C	Musculocutaneous vessel that penetrates the fascia	Transverse rectus abdominis myocutaneous flap

Perforator Flaps

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The concept of perforator flaps emerged from the postulate that a single perforator artery could vascularize a definite skin territory and its subcutaneous component without having to include the adjacent muscular tissue. The first reported perforator flap was described by Koshima in 1989 in which an abdominal skin paddle was harvested without the rectus abdominis muscle, including only the skin and adipose tissue, and was vascularized by a single perforator of the deep inferior epigastric artery.[12] This flap is now known as the *deep inferior epigastric perforator* (DIEP) flap.

Numerous perforator flaps have since been described. When an artery is dissected from its arborization into the skin to its proximal origin, it is termed a perforator flap. Perforator flaps differ from conventional musculocutaneous or fasciocutaneous flaps by way of their anatomic origin. Skin and subcutaneous tissue are supplied by the following five vascular plexuses: subepidermal, subdermal, dermal, subcutaneous, and fascial (sub- and suprafascial). Conventional flaps originate to vessels beneath muscle layers or within the fascia. In contrast, perforator flaps are supplied by the more distal subcutaneous or subdermal vessels, which allows for elevation of just the skin paddle without the requirement of other tissues.[13]

In an attempt to define and classify perforator flaps, Blondeel et al. defined a perforator flap as the following: "A perforating vessel, or, in short, a perforator, is a vessel that has its origin in one of the axial vessels of the body and that passes through certain structural elements of the body, besides interstitial connective tissue and fat, before reaching the subcutaneous fat layer."[14] Some authors make a distinction between a direct (only goes through fascia before reaching skin) and an indirect perforator (goes through other tissues before reaching skin).[15] In 2003, the Gent consensus was published proposing a classification and a nomenclature scheme (**TABLE 9-3** and **FIGURE 9-9**).[14]

FIGURE 9-9

The Gent classification illustrated (Used with permission from Blondeel PN, Van Landuyt KHI, Monstrey SJM, et al. The "Gent" consensus on perforator flap terminology: preliminary definitions. *Plast Reconstr Surg*. 2003;112(5):1378–1383. https://journals-lww-com.ezproxy.cihe.edu.hk/plasreconsurg/Abstract/2003/10000/The__Gent__Consensus_on_Perforator_Flap.24.aspx.)

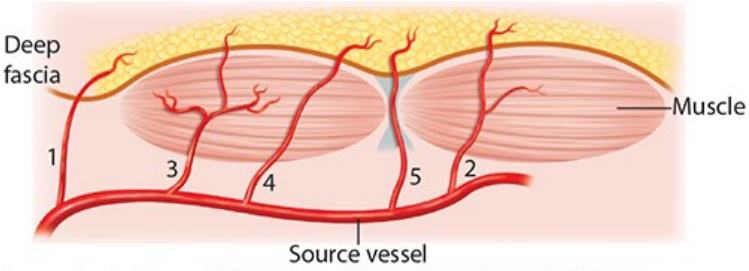

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TABLE 9-3

Gent Classification of Perforator Flaps

Type 1: Direct perforators perforate the deep fascia only. The perforator artery runs from its main vessel directly to the deep fascia.
Type 2: Indirect muscle perforators predominantly supply the subcutaneous tissues. A perforator travels through a muscle but mainly vascularizes a skin territory; few branches are going into the muscle.
Type 3: Indirect muscle perforators predominantly supply the muscle but have secondary branches to the subcutaneous tissues.
Type 4: Indirect perimysial perforators travel within the perimysium between muscle fibers before piercing the deep fascia.
Type 5: Indirect septal perforators travel through the intermuscular septum before piercing the deep fascia.

This classification was simplified in 2003 with only three types of flaps included (**TABLE 9-4** and **FIGURE 9-10**).[16] Despite these efforts, controversy still exists among the definition of perforator flaps, and there have been calls to refine the terminology.[2,17]

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FIGURE 9-10

Illustration of the three types of perforator flaps

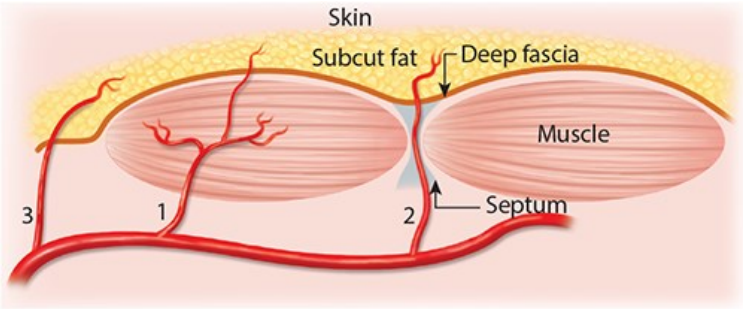

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TABLE 9-4

Simplified Classification of Perforator Flaps

Type 1: Indirect "muscle" or myocutaneous perforators traverse through muscle to pierce the outer layer of the deep fascia to supply skin.
Type 2: Indirect "septal" or septocutaneous perforators traverse only through a septum to reach the deep fascia and the skin.
Type 3: "Direct" perforators perforate the deep fascia only. (From Blondeel PN, Van Landuyt K, Hamdi M, Monstrey SJ. Perforator flap terminology: update 2002. *Clin Plast Surg.* 2003;30(3):343–346.)

To standardize nomenclature of perforator flaps, the Gent consensus proposed that "A perforator flap should be named after the nutrient artery or vessels and not after the underlying muscle. If there is a potential to harvest multiple perforator flaps from one vessel, the name of each flap should be based on its anatomical region or muscle."[14] For example, a perforator flap based on the thoracodorsal vessels, the main vessel for the latissimus dorsi, should be labeled a *thoracodorsal artery perforator flap* (TAP flap).

Since their discovery, over 350 perforators have been identified. Perforator flaps can be elevated as pedicled or free flaps.[18] In 2009 Saint-Cyr et al. introduced the term "perforasome" to describe the cutaneous territories supplied by various perforator vessels (**FIGURE 9-11**).[19] The authors mapped and compiled the perforators and their associated tissues via injection and imaging. This concept is similar to the term angiosome which describes the vascular territory supplied by each artery. (More information on angiosomes follows.)

FIGURE 9-11

Common perforasomes and flow direction (Used with permission from Saint-Cyr, M., Wong, C., Schaverien, M., Mojallal, A. & Rohrich, R. J. The perforasome theory: vascular anatomy and clinical implications. *Plast Reconstr Surg.* 2009;124:1529–1544. https://journals-lww-com.ezproxy.cihe.edu.hk/plasreconsurg/Abstract/2009/11000/The_Perforasome_Theory__Vascular_Anatomy_and.20.aspx.)

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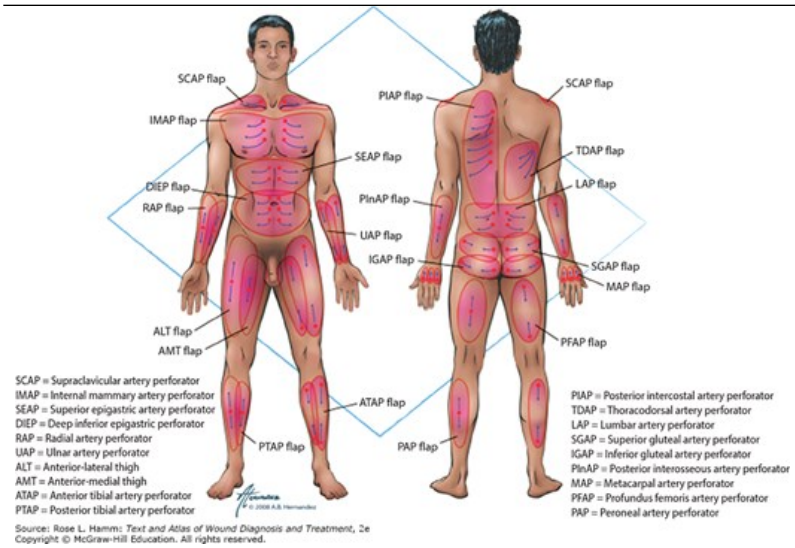
SCAP flap
IMAP flap
SEAP flap
DIEP flap
RAP flap
UAP flap
IGAP flap
ALT flap
AMT flap
ATAP flap
PTAP flap
PIAP flap
SCAP flap
TDAP flap
LAP flap
PInAP flap
SGAP flap
MAP flap
PFAP flap
PAP flap
SCAP = Supraclavicular artery perforator
IMAP = Internal mammary artery perforator
SEAP = Superior epigastric artery perforator
DIEP = Deep inferior epigastric perforator
RAP = Radial artery perforator
UAP = Ulnar artery perforator
ALT = Anterior-lateral thigh
AMT = Anterior-medial thigh
ATAP = Anterior tibial artery perforator
PTAP = Posterior tibial artery perforator
PIAP = Posterior intercostal artery perforator
TDAP = Thoracodorsal artery perforator
LAP = Lumbar artery perforator
SGAP = Superior gluteal artery perforator
IGAP = Inferior gluteal artery perforator
PInAP = Posterior interosseous artery perforator
MAP = Metacarpal artery perforator
PFAP = Profundus femoris artery perforator
PAP = Peroneal artery perforator
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Perforator flaps have become a mainstay of reconstruction. The DIEP flap is now the preferred technique in breast reconstruction as it offers excellent aesthetic outcomes and decreased donor site morbidity compared to previous techniques such as the transverse rectus abdominis myocutaneous (TRAM) flaps.[20] Other techniques such as profunda artery perforator (PAP), thoracodorsal artery perforator (TDAP), and superior gluteal artery perforator (SGAP) flaps have been described and are now commonly used for a variety of reconstructive purposes, including but not limited to head and neck reconstruction, abdominal wall reconstruction, and extremity reconstruction.[21,22]

The perforator flap offers many advantages over the conventional myocutaneous flap. The wealth of perforators provides an abundance of donor sites. Perforator flaps do not require elevation of the underlying muscle; therefore, the muscle can be spared and donor site function is preserved, as opposed to myocutaneous or myofascial flaps. Unlike conventional flaps, perforator flaps can be oriented in any direction on the perforator, which allows for optimal defect coverage. Perforator flaps can be much more versatile than conventional flaps because they can be readily thinned and shaped to conform to the defect, they can be oriented freely based on the pedicle, and they have a longer pedicle.[23] These properties may stem from the observed phenomenon that pedicled flaps are hyperperfused compared to normal circulation.[24] However, these benefits do not come without a cost. Perforator flaps often require tedious, time-consuming dissection, which can prolong operative time. In addition, the distribution of perforator vessels is less consistent than larger vessels, necessitating more preoperative studies and intraoperative effort.[25]

Perforator flaps are a powerful tool in the reconstructive surgeon's armamentarium. The increase in popularity of these flaps has been supported by improved understanding, which has enabled perforator flaps to supplant many conventional flaps. The mantra of “flap of choice” is slowly fading away as the versatility of perforator flaps continues to evolve and expand.[26] Continued study is needed to better characterize and define the vascular distribution, and future investigation will refine the use of these flaps as well as potentially discover their new applications.

Propeller Flaps

Since its inception in 1991, the propeller flap has undergone a series of transformations to maximize tissue coverage and improve its use in soft-tissue defect reconstruction throughout the body. The original flap described by Hyakusoku et al. was adipocutaneous in nature and consisted of a skin island with two “propeller blades” at each end of a vascular pedicle.[27] Much like a propeller, the flap was then rotated 90° around its pedicle for the release of scar contractures at the axilla and elbow. A modified version of the original propeller flap was later described by Hallock and included two major changes: as skeletonized perforating vessel and a flap rotation angle of 180°.[28]

The different types of propeller flaps commonly used in current practice are described in a consensus by the Advisory Panel of the First Tokyo Meeting on Perforator and Propeller Flaps.[29] The “Tokyo” consensus, analogous to the “Gent” consensus on perforator flaps, describes propeller flaps as an “island flap that reaches the recipient site through an axial rotation.”[30] This definition excludes flaps whose principal movement is advancement and flaps that are not designed as a complete island.

The “Tokyo” consensus also sets forth how to classify propeller flaps.[29] Proper terminology necessitates identifying the type of nourishing pedicle, the

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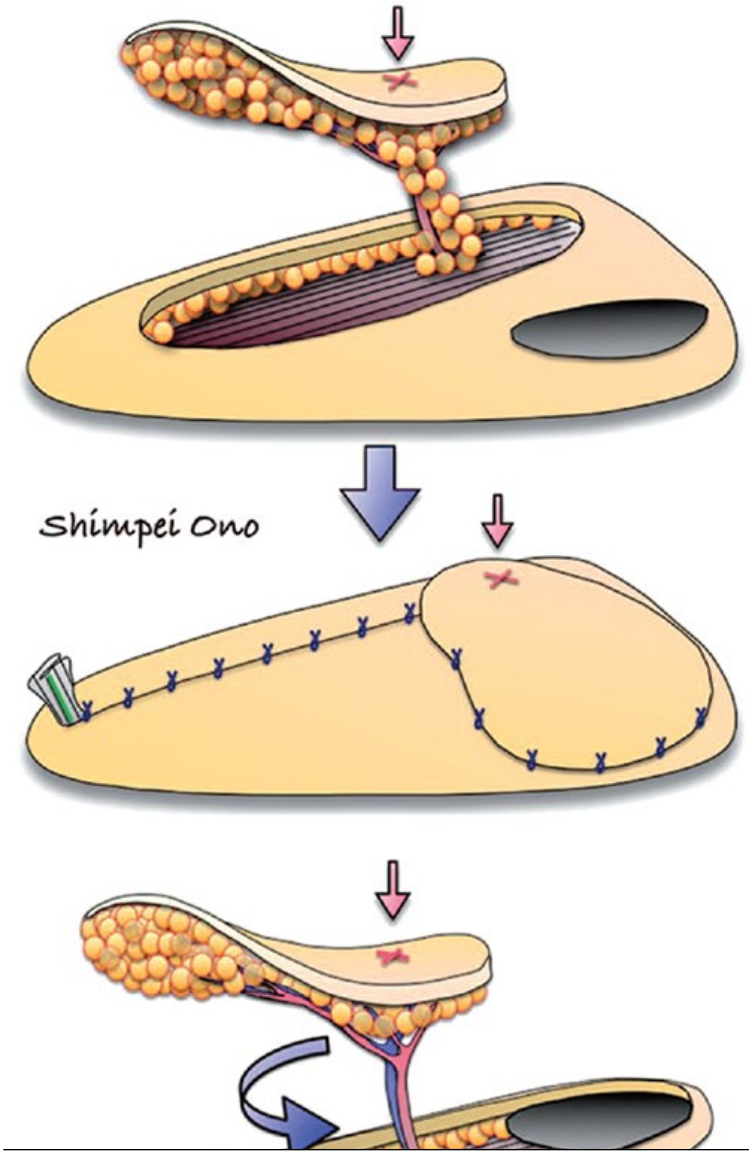
degree of skin island rotation, and the artery of origin of the perforator vessel, if known. The three following types of propeller flaps are distinguished based on the type of nourishing pedicle: (1) The *subcutaneous pedicled propeller flap* is analogous to the original propeller flap described in 1991, where a subcutaneous pedicle of unknown vascular origin is rotated 90° without the need to visualize or isolate perforator vessels, followed by direct closure of the donor site. (2) The *perforator pedicled propeller flap* incorporates the perforator flap concept, leading to a propeller flap supplied by a perforator vessel that determines the borders of the skin island overlying it. This perforator vessel is visualized, dissected, and skeletonized, allowing for a rotation up to 180° as opposed to the original subcutaneous propeller flap. Freeing the vessel from fascial adhesions minimizes the risk of vessel buckling during flap rotation. The greater range of rotation inherent in the pedicled propeller flap design makes it the most commonly used type of propeller flap. (3) The *supercharged propeller flap* is a modified version of the pedicled propeller flap that allows for greater tissue coverage. Its design involves the dissection and anastomosis of an extra vascular pedicle to enhance arterial inflow, venous outflow, or both. As such, it is used if extra flap length is needed that would not be sufficiently supplied by a single perforator vessel, or for salvage procedures (**FIGURE 9-12** and **TABLE 9-5**).

FIGURE 9-12

Illustration of the three types of propeller flaps Subcutaneous pedicled propeller (*top*), perforator pedicled propeller (*middle*), supercharged propeller (*bottom*). (Used with permission from Pignatti M, Ogawa R, Hallock GG, et al. The "Tokyo" consensus on propeller flaps. *Plast Reconstr Surg.* 2011;127(2):716–722. https://journals-lww-com.ezproxy.cihe.edu.hk/plasreconsurg/Abstract/2011/02000/The__Tokyo__Consensus_on_Propeller_Flaps.27.aspx.)



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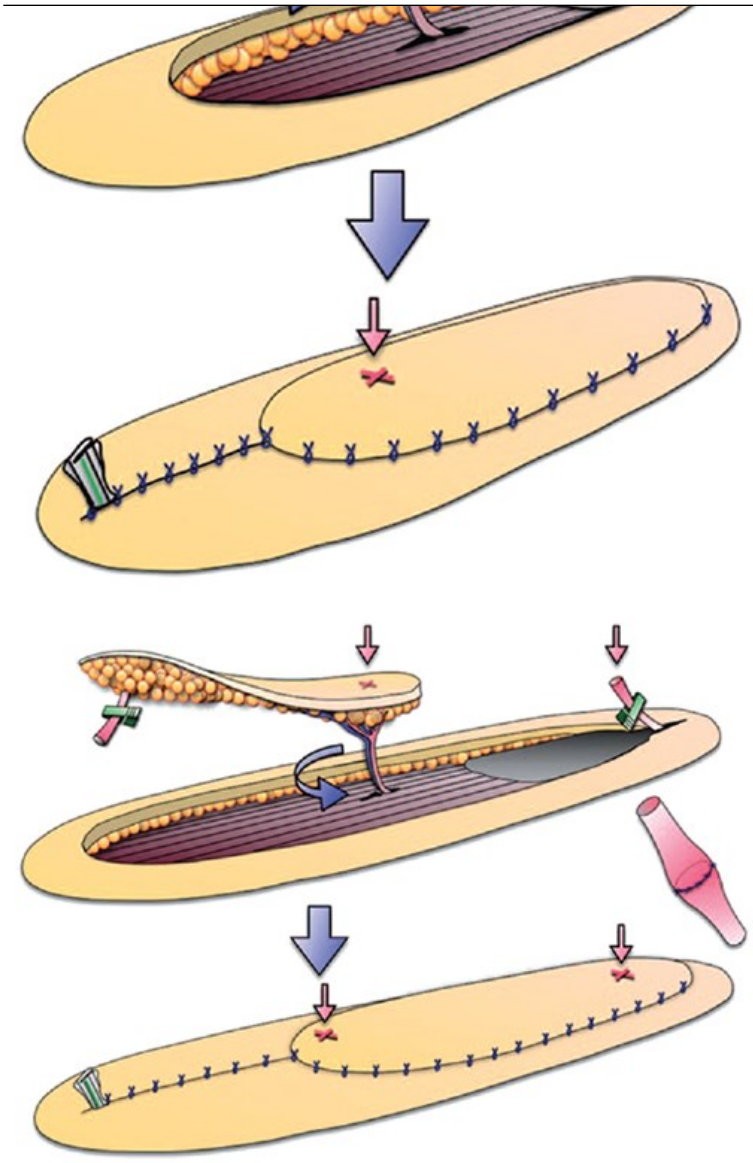

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TABLE 9-5

Propeller Flap Types and Their Characteristics

Flap Type	Characteristics	Technique
Subcutaneous pedicled propeller	Random subcutaneous pedicle of uncertain origin, up to 90° rotation	Macroscopic (vessel not visualized)
Perforator pedicled propeller	Skeletonized perforating vessel, origin known from anatomy or directly visualized, up to 180° rotation	Magnification needed (vessel visualized and dissected)
Supercharged propeller	Skeletonized perforating vessel plus extra vein, artery or both, origin known from anatomy or directly visualized, up to 180° rotation	Microsurgical (vessel visualized and dissected, plus microsurgical anastomosis)

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An updated "Tokyo" consensus was proposed by Toia et al. in 2017, whereby a new type of propeller flap is described that does not fit into the established classification. The newly proposed *axial pedicled propeller flap* is by definition a propeller flap since it involves an island of tissue that reaches its recipient site through axial rotation; however, its pedicle is neither subcutaneous nor perforator, but rather comes out of the bone to enter the flap perpendicularly.[31] Two such types of flaps have been reported in the literature: the supratrochlear artery axial propeller (STAAP) flap and the deep lingual artery axial propeller (DLAAP) flap.[31]

Propeller flaps are an attractive reconstructive option due to their versatility. Reconstruction can be performed in the upper and lower extremities, the head and neck, the trunk, abdomen, and buttocks. In order to be successful, the defect should be in a well-vascularized territory, be small to medium sized, and be surrounded by healthy tissue.[32]

There are many advantages to using propeller flaps, which set it apart from other techniques. The most obvious benefit is the great mobility, which can reach 180° of rotation, allowing for maximum utilization of the flap. The design of the flap allows for partial coverage of the secondary defect (often by primary closure) and scar manipulation, which is especially beneficial in cosmetically sensitive areas. Another advantage of propeller flaps is their ability to exceed the traditional 3:1 length to width ratio; in fact, successful flaps with a ratio of greater than 6:1 have been reported.[33] This may be due in part to the ability of perforator vessels to adequately perfuse beyond their angiosome/perforasome territory into those of their neighbors.[34] In all, these flaps are able to offer robust coverage with skin that offers similar quality and type, which can be used in a variety of applications.[35]

No flap is perfect, and propeller flaps do have disadvantages that must be carefully considered when they are used. The complication rate can be as high as 31% in lower extremity reconstruction due to the lack of well-vascularized recipient tissue. Certain demographics such as infants and the elderly have higher rates of complications as well.[32] As stated, these flaps cannot cover as great of an area as can others, due to being elevated on a single pedicle. Rotation of the flap can cause undue tension on the pedicle and the donor site, which must be carefully monitored. Finally, as with other flap modalities, the dissection can be arduous, as perforator anatomy can be varied.[32]

Propeller flaps, when properly utilized, can be an excellent reconstructive option that may become more prevalent as they are continually studied. Although propeller flaps have lower complications than conventional pedicled flaps, there are currently few studies analyzing risk variables and propeller flap complications such as venous congestion, superficial necrosis, and flap failure.[36,37] Consistent risk variable documentation by providers and future multicenter studies will help identify what patient factors and flap characteristics are more likely to result in success or complications.

The Angiosome Concept and the Choke Vessels

In 1987, Taylor and Palmer defined the concept of an *angiosome*. They described the skin perfusion as a 3D jigsaw puzzle where each artery originating from a source vessel perfuses a skin territory with its underlying structure.[38] For example, the deep inferior epigastric system vascularizes a skin territory labeled the deep inferior epigastric angiosome. Each defined artery supplying the skin has its own angiosome.

Angiosomes are not isolated entities and are related to each other by connecting vessels called *choke vessels*. These vessels are at the margin of adjacent angiosomes and can be more or less open to allow perfusion from one territory to another. This concept will be later explained in the delay phenomenon section.

Veins and Venosomes

The venous system is composed of a deep and a superficial system, connected by perforator veins. An artery may have one or two veins with a similar anatomical pathway. Such a vein is labeled *vena comitans* or *venae comitantes* in the plural form. (*Comitans* comes from Latin and means to accompany.) For example, a radial forearm flap may have the cephalic vein as a superficial venous drainage system as well as two *venae comitantes* along the radial artery. These two systems ultimately join near the elbow via a communicating vein. Of note, some arteries do not have *vena comitans*. For example, the carotid artery does not have any accompanying vessels per se.

Using the same concept as angiosomes, Taylor et al. described a comparable venosome concept. A close relationship was found between the arterial and venous territories of each muscle.[38] Instead of having choke vessels on the periphery of their territories, venosomes have *oscillating veins* that have the unique characteristic of no valves. In contrast, venae comitantes have valves to direct blood flow toward the source artery.[39]

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Physiology

The Vascular Sequence Following Flap Harvest

The elevation of a flap has a major impact on local blood flow. The angiosome concept explains the interconnection between different vascular territories. During a flap harvest, blood inflow and outflow are often restricted to a single pedicle; the numerous small connections between the flap and its surrounding tissues are severed. Microcirculation undergoes a reorganization to allow adequate blood inflow and outflow; this process lasts up to 4 weeks. The sequence for pedicle flaps can be described as follows[40]:

0–24 hours: The immediate response of the microcirculation is vasospasm attributed to a local axon reflex phenomenon and hyperemia caused by hypoxia and loss of sympathetic control. The overall effect is a decrease in blood flow. There is a decrease in circulation during the initial 6 hours, reaching a plateau at 6–12 hours postharvest, followed by a progressive increase at 12 hours postharvest. Congestion and edema are frequently observed during this post-surgical period.

1–3 days: An improvement in pulse amplitude is noted when spontaneous vascular tone returns around the third day. There is an increase in the number and caliber of longitudinal anastomoses as well as the number of small vessels in the pedicle. *It is imperative for flap survival to avoid shear and congestion during this period.*

3–7 days: Circulation increases progressively with a plateau on day 7. Anastomoses noted earlier become significantly functional at 5–7 days, and there is an increase in both vessel size and number.

1 week: Blood circulation is well established between pedicle and recipient bed. Pulse amplitude approaches preoperative levels.

7–14 days: No further significant increase in vascularization occurs and maximum inflow is achieved. Circulation efficiency is superior to preoperative levels during the period of 10–21 days.

2 weeks: There is a progressive regression of the vascular system. Maturation of the new anastomoses between flap and recipient bed takes place.

3 weeks: The vascular pattern is similar to preoperative levels. Vascular connections between the pedicle flap and the recipient bed are fully developed.

4 weeks: There is a decrease in diameter of all vessels, including preexisting vessels. Circulation efficiency is normal and skin color becomes more normal without the increased vascularity.

The sequence that follows a free flap harvest and anastomosis is similar but retains a major distinction. When blood vessels are sectioned, they lose the direct nervous regulation by sympathetic and parasympathetic fibers. However, the re-establishment of circulation between the flap and its recipient bed shares similitudes.

Microcirculation Regulation

Local blood flow in the microcirculation is regulated by multiple factors, each one ultimately causing vasoconstriction or vasodilation. The summation of these opposing forces eventually determines the final local blood flow. In the case of a flap in which microvascular rearrangements are multiple secondary to flap dissection, these factors are of paramount importance. They must be carefully evaluated and controlled to allow flap survival. These factors can be classified as being regulated systemically or locally.

Systemic Regulation

Systemic regulation has two components: neural regulation and humoral regulation. *Neural regulation* is the dominant effect of systemic regulation and is mostly via sympathetic adrenergic fibers. Vasoconstriction is induced by α-adrenergic receptors while vasodilation is the result of β-adrenergic receptors. Other fibers have also been implicated in the overall vascular tone. Serotoninergic fibers induce vasoconstriction while parasympathetic cholinergic fibers, via muscarinic receptors, cause vasodilation. The combination of all these forces ultimately determines the neural basal tone of vascular smooth muscle fibers in blood vessel.[41]

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Humoral regulation is controlled by circulating hormones. The most important are epinephrine and norepinephrine acting on α-adrenergic receptors, thereby causing vasoconstriction. Serotonin, thromboxane A_2, and prostaglandin $F_{2\alpha}$ also cause vasoconstriction. Substances causing vasodilation are prostaglandin E_1, prostaglandin I_2, histamine, bradykinin, and leukotrienes C_4 and D_4.[41]

Local Regulation

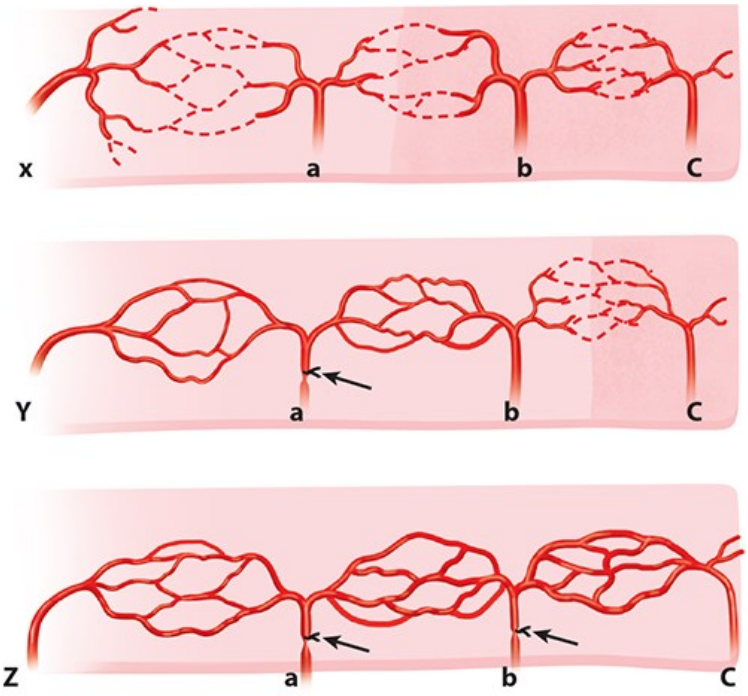
Local regulation is also known as autoregulation and is divided into two separate components. The first local control is exercised through metabolic factors; for example, hypercapnia, hypoxia, and acidosis cause vasodilation. The second component relates to physical factors. Increased tissue perfusion pressure can initiate a myogenic reflex that causes vasoconstriction in order to maintain constant capillary blood flow. Hypothermia causes vasoconstriction via stimulation of vascular smooth muscles. Hyperthermia causes vasodilation by the opposite effect. Finally, it has been speculated that blood rheology, defined as the study of blood flow, can affect perfusion. High blood viscosity can diminish flap perfusion and viability.[41]

Delay Phenomenon

The final design of a flap is limited by the previously described vascular territories; however, the flap dimension can be extended using a delay procedure (**FIGURE 9-13**). In physiological conditions, each angiosome has a peripheral area connecting to other angiosomes through choke vessels. These vessels are not aligned in a specific direction and are of small caliber.

FIGURE 9-13

The delay phenomenon for a flap procedure Surgical delay of a flap induces vasodilation of choke vessels, leading to increased perfusion and venous drainage of the distal portion of a flap. The shaded area in flap "X" represents distal necrosis. After surgical delay or vascular bundle "a" in flap "Y", there is increased distal flap perfusion secondary to changes in choke vessels between "a" and "b" but necrosis in the distal tip because vessel "b" was not delayed. In flap "Z" both vascular bundles "a" and "b" have been delayed, thus leading to increased perfusion via choke vessels between both a/b and b/c segments. Maximal surgical delay in flap "Z" leads to the most distal flap survival.



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In a delay procedure, only a portion of a flap is elevated and then sutured back in place for 2–3 weeks. The goal is to "induce a level of ischemia in the distal portion of the flap that does not cause necrosis yet conditions the tissue so that the flap will survive after later elevation."[42] In this chapter, we previously described the vascular sequence following flap harvest that indicates the blood flow is near normal or normal after 2–3 weeks. For this

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reason, final flap harvest is usually performed between 2 and 3 weeks after the initial procedure.

The results of a delay procedure are multiple. (1) There is an opening of choke vessels that are normally closed. (2) There is a reorientation of the flap vessels in a more longitudinal pattern. (3) There is a sprouting of new vessels within the flap via angiogenesis. (4) The physiologic stress of the delay procedure increases the vessel caliber.[43]

As an example, the deltopectoral fasciocutaneous flap, initially described by Bakamjan, is frequently used for head and neck reconstruction and can be extended using a delay procedure. The original design, based on internal mammary artery perforators, can only extend up to the medial border of the deltoid muscle. This anatomic consideration significantly limits the reach of the flap. Using a delay procedure, the skin paddle can be extended to the lateral aspect of the deltoid muscle. During the initial procedure, the distal skin paddle covering the deltoid is elevated but sutured back in place; however, the base of the flap is not elevated. Three weeks later, the entire skin paddle is elevated.

Tissue Expansion

Tissue expansion may be used for skin deficits where a specific color match or texture of skin is desired for reconstruction (**FIGURE 9-14**). For example, in a burned patient, scalp skin expansion may be employed to reconstruct deficits of hair bearing skin. In tissue expansion, an expander device is inserted subcutaneously and after a week to 10 days of acclimation is gradually inflated (eg, once a week for 6 weeks) so that a force stretches the targeted tissue over weeks to months prior to transfer. Tissue expanders are used for a variety of applications, the most common of which are breast reconstruction and repair of scalp defects.

FIGURE 9-14

Examples of tissue expansionA. Breast expansion with tissue expanders placed under the pectoralis muscle. **B.** Scalp expansion with tissue expanders placed under the scalp.

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The tissue to be expanded initially undergoes mechanical creep, which is comprised of realignment of collagen fibers and displacement of water from the ground substance. Chronic distension initiates stretch responses (collectively termed biological creep) that include increased rates of mitosis, upregulated angiogenesis, and new tissue production.[44] The final result is a mixture of tissue stretching and growth.

The device, generally a silicone envelope that is progressively inflated with saline, creates a capsule around itself and causes specific changes to the skin and the underlying tissues. The capsule is composed of dense fibrous fibers and becomes less cellular over time, reaching maximal thickness after

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2 months of expansion. Fibroproliferation and capsular contracture are potential complications that are associated with radiation therapy.[45]

During expansion of the skin, the epidermis initially increases in thickness while the dermis and the subcutaneous adipose tissue are significantly thinner after expansion.[46] Skin appendages are not histologically affected but develop decreased density, and the muscle atrophies.[47] An expander compressing a bone causes a decrease in bone thickness and volume but does not affect bone density, for example, during a scalp expansion. At the periphery of the expander, an increase in bone thickness and volume is noted.[48] Research has shown that many of these changes are reversible. The capsule resolves after expander removal and little histological evidence persists after 2 years following the removal of the device. Similarly, dermis and subcutaneous tissue regain thickness after expander removal.[47]

Tissue expanders come in three varieties: round, rectangular, and crescentic. van Rappard et al. calculated that rectangular devices provide the largest increase in surface area, 38% expansion compared to 32% of cresenteric and 25% of round.[49] Various claims have been reported regarding the required width of the expander relative to the defect, but most authors claim a requirement of 2.5–3 times the diameter of the defect.[50]

Expanded flaps provide several advantages over unmodified ones. The most obvious advantage is increased coverage of defects with the same donor footprint. Coverage can be achieved via advancement or transposition.[51] As previously stated, tissue expansion can provide matching skin tone, texture, and hair patterns, which are especially valuable in aesthetically sensitive areas. Expanded tissue can be closed primarily, which can help minimize donor site morbidity. Since the entire sequence of tissue expansion is planned, scar placement and size can be manipulated to be minimally disruptive. Finally, tissue expansion is a reliable and well-understood technique which can be utilized with known results.

As with any surgical technique, tissue expansion is not without disadvantages. At least two operations are required for tissue expansion, one to implant and one to remove the expander. Reconstruction must be delayed for the duration of the expansion process, which, especially in aesthetically sensitive areas such as the head and neck, can be very disruptive to a patient's quality of life. The implant creates a dramatic and instantly noticeable deformity that the patient must be prepared to face.

Complications of tissue expander use can delay or prevent reconstruction. Minor complications include hematoma or seroma formation, exposure of the valve, or insufficient expansion; major complications include infection, exposure of the implant, implant deflation, or flap necrosis. Lower extremity expansion confers the highest risk, which may be secondary to anatomic limitations and decreased vascularity of the tissue, as compared to the head, neck, or trunk.[50]

Monitoring

The ultimate success of any flap transfer is not measured immediately after surgery but only after adequate *healing and remodeling* of the flap have occurred. As noted earlier, it takes several days to re-establish satisfactory blood supply and, therefore, ensure flap survival. The first 5–7 days are critical because the entire flap relies on its vascular pedicle for blood supply. At 1-week post-surgery, additional blood supply has been re-established, consequently decreasing the overall dependency from the main pedicle. However, flaps have been lost later than 7 days postoperatively, illustrating the variability of flap healing. Since flap salvage procedures are available and can be successful, appropriate monitoring is essential to detect early signs of flap insufficiency.

The monitoring of a flap can be performed in various ways. The most common include clinical parameters such as color, temperature, and turgor. These metrics are very sensitive for flap danger to the point that some authors question the need for any other monitoring modality.[52] That stated, invasive monitoring techniques such as an implantable Doppler flow probe and transcutaneous oxygen saturation probe are available to the clinician and can be useful for buried flaps without skin paddles. Implantable Doppler has been shown to improve postoperative monitoring of at-risk flaps, which can allow for more prompt response and salvage of the flap.[53] More recently, modalities (eg, near-infrared spectroscopy with contrast agents such as indocyanine green) have provided additional mechanisms for measuring flap perfusion.[54]

Physiological Parameters

Vital Signs

As with the evaluation of any specific medical condition, vital signs are of critical importance when monitoring a patient with a flap. Normal circulation

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is influenced by temperature, blood pressure (BP), heart rate (HR), and/or respiratory rate (RR). For a flap where the only vascular supply is limited to a single artery and one or two veins, slight variations of these parameters can have a significant influence on both arterial and venous blood flow.

As discussed earlier, hypothermia can lead to vasoconstriction and is a cause of significant blood redistribution in the body. For example, a flap on a lower extremity may suffer from vasoconstriction if the body needs to conserve heat and maintain normal core body temperature. As a consequence, vasoconstriction in a flap can lead to transient ischemia and induce a cascade of events that leads to flap failure.

The importance of maintaining adequate systemic blood pressure for flap perfusion is straightforward and because flaps have relatively small conduits for arterial inflow and venous outflow, periods of systemic hypotension are poorly tolerated. Respiratory rate can also influence flap survival. By acting on blood pH, RR can affect the overall pH. Hypercapnia, hypoxia, and acidosis cause vasodilation and have a direct effect on flap tissue perfusion.

Capillary Refill Time (CRT)

Normal skin perfusion fills the capillaries in the dermal layer of the skin. By simple digital pressure, these capillaries can be emptied and will again refill. The time needed to refill capillaries is directly related to the local arterial and venous pressures; normal CRT is less than 2 seconds.[55,56] When monitoring a flap, a CRT between 1 and 3 seconds is considered normal. If CRT is 1 second or less, it is an indicator of *venous congestion*.[16] This phenomenon may be explained by the following: Since perfusion pressure on the arterial capillary side is two times the pressure of the venous capillary,[57] digital pressure on the skin forces blood to the venous side of the capillaries. If venous drainage is impaired, venous capillary blood pressure will increase and blood emptying will be limited by the increased venous blood pressure. Thus, CRT is then faster than expected in venous congested flaps. However, a CRT more than 3 seconds is a sign of arterial insufficiency. A prolonged CRT can be explained by the fact that when arterial capillary pressure is lower than normal, the time to refill capillaries is longer (**FIGURE 9-15**).

FIGURE 9-15

Capillary bed pressures The capillary bed has a pressure of 32 mmHg on the arterial side and 15 mmHg on the venous side. If there is venous congestion in a flap, pressure on the skin does not force fluid into the venules; therefore, the capillary refill time is less than normal. In contrast, if the arterial flow is impaired, refill time will be longer than the normally expected 2–3 seconds. This screening test can be used to monitor vascularization following flap surgery and to detect early perfusion impairments. (Used with permission from Moris SF, Taylor GI. Vascular territories. In: Neligan PC, ed. *Plastic Surgery*. 3rd ed. Philadelphia, PA: Elsevier; 2013:480.)

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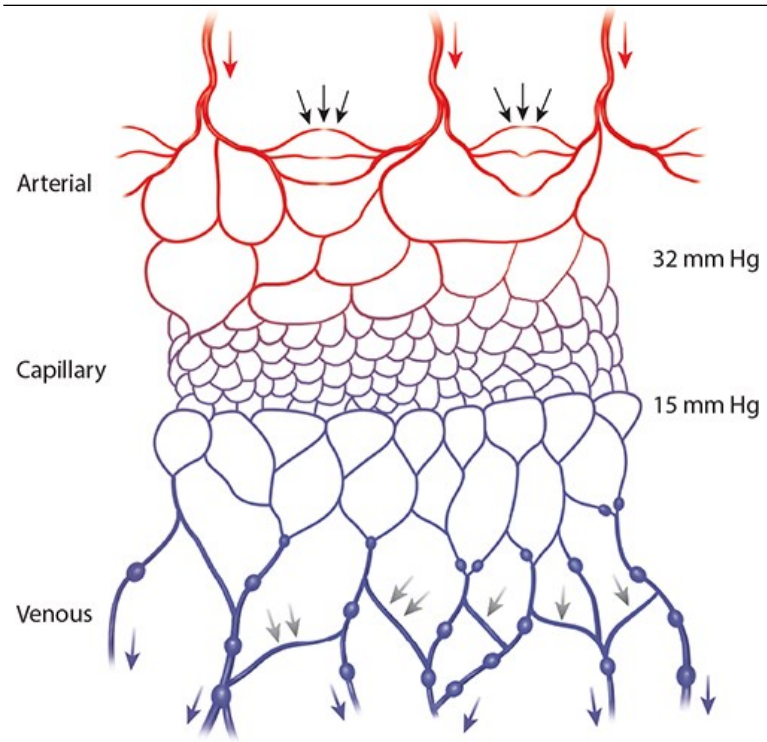

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Color

Skin color of a flap can be readily monitored for viability (**FIGURE 9-16**). Depending on the patient's skin complexion, pale or dusky skin can be assessed in a few seconds. Pallor is associated with underperfusion while pink, bluish, or dusky skin is associated with venous congestion. Skin transferred from one region of the body to another may not have the same basal color. For example, a TRAM flap for breast reconstruction can have a much lighter skin paddle than the chest wall skin. In this situation, a baseline assessment of skin color (ie, digital photograph) is recommended for use as a reference for postoperative monitoring.

FIGURE 9-16

Color monitoring for flap viability Congested purple skin in a free radial forearm flap for ocular exenteration. The purple color is indicative of venous congestion. This situation warrants surgical re-exploration but in some cases is treated with leech therapy to relieve the congestion temporarily until capillary ingrowth is reestablished.

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Flap Temperature

Any flap, including a skin flap, can be evaluated for temperature. An abnormal temperature is a symptom of inadequate perfusion. This examination is performed subjectively with the dorsum of the hand or objectively with an infrared skin thermometer, comparing the temperature of the flap with its surrounding environment. A cool flap compared to its adjacent tissue is a sign of hypoperfusion. Flap hyperthermia is less of a concern and hard to evaluate clinically as flaps are often externally warmed in the immediate postop period to reduce vasoconstriction.

Needle Prick

A simple method to assess perfusion is by creating a small puncture wound on the flap using a 21-gauge needle. Whereas a healthy flap would exude bright red blood, the absence of blood can be interpreted as arterial insufficiency while dark cyanotic blood would be a sign of venous congestion.

Ancillary Monitoring Methods

In situations where clinical parameters are not available (eg, buried flap) or not reliable (eg, on a darker skin patient), more objective measurement tools are utilized in an effort to better assess flap viability.

Doppler

Since flaps are based on a vascular pedicle, blood flow can be evaluated by ultrasonography using a pencil-type Doppler probe, which measures the velocity of the blood flow in the artery and vein (**FIGURE 9-17**). Arterial flow is classically triphasic while venous flow is monophasic. Unfortunately, a Doppler signal is not always available because of flap configuration and the signal quality can vary; however, the Doppler study is reliable when it is audible. The loss of a previously good signal is correlated with flap failure, although the rate of false-positive loss of a signal may be important.[58] Sometimes, it can be difficult to evaluate the appropriate artery, for example, in a patient with a partially missing cranial vault after a cranioplasty where a latissimus dorsi flap is used to cover the deficit. The recipient vessels chosen are often the superficial temporal artery and veins. Without the bony structure covering the brain, a positive Doppler signal from the cerebral circulation can be mistakenly assessed as a positive flap pedicle signal. For this reason, more sophisticated methods have been developed, including an implantable Doppler probe directly around the vessels.

FIGURE 9-17

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A handheld Doppler The pencil-style handheld Doppler is used to monitor blood flow in the pedicle artery and to detect any decrease in flow that may compromise flap perfusion.

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Transcutaneous Oximetry

Another method of evaluating flap perfusion is through pulse oximetry. Pulse oximetry, which is based on absorption of a specific wavelength of light by the patient's hemoglobin, is a reliable indicator of arterial perfusion. In the case of a replanted finger, a simple pulse oximetry sensor can be placed around the tip of the finger to get constant saturation measurement. For flaps with skin, transcutaneous probes, which can be affixed with adhesive tape, have been developed to measure flap saturation. As with the Doppler signal, a sudden change in saturation, as opposed to the absolute value of the saturation, is an indicator of potential flap failure.

Contrast agents for assessment of tissue perfusion

Recently, indocyanine green (ICG) dye has been utilized to directly visualize flap perfusion (**FIGURE 9-18**). When injected into the bloodstream, it tightly binds to plasma proteins and is, therefore, confined to the vascular system. A laser is used to detect the dye, which has a peak spectral

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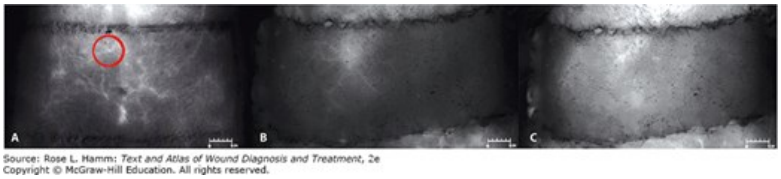
absorption at 800 nm. Since ICG dye has a short half-life of 3–4 minutes, it quickly enters vascularized tissue flaps but then also rapidly washes out prior to being metabolized by the liver. For example, after a mastectomy, random skin flaps that have questionable perfusion can be assessed by ICG angiography intraoperatively when perfusion is not always clinically obvious. Real-time data can be used to guide excision of marginally perfused tissue.[59,60]

FIGURE 9-18

Indocyanine green staining of a breast flap Indocyanine green angiography is used to monitor flap perfusion in an animal study involving abdominal perforator flaps. **A.** A preoperative view of the target perforator. **B.** Early injection showing localization of the perforator. **C.** Late injection phase with hyperlucent and hyperopaque zones. (Used with permission from Monahan J, Hwang BH, Kennedy JM, Chen W, Nguyen GK, Schooler WG, Wong AK. Determination of a perfusion threshold in experimental flap surgery using indocyanine green angiography. Ann Plast Surg. 2014;73(5):602–604. https://journals-lww-com.ezproxy.cihe.edu.hk/annalsplasticsurgery/Abstract/2014/11000/Determination_of_a_Perfusion_Threshold_in.26.aspx.)



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Fluorescein dye was used for many years as an indicator of flap arterial perfusion prior to the development of ICG angiography. The presence of the dye is assessed with a Wood lamp after the dye is systemically injected in a manner similar to ICG dye. However, the absence of dye is not always associated with insufficient perfusion (eg, less reliable). Since this agent often overestimates nonperfused tissue, it is rarely used in the authors' practice.

Complications

Flap surgery, as with any other type of surgery, can have complications. Besides the usual complications associated with a surgical procedure under general anesthesia, unique difficulties are specific to flap procedures. Complications can be to the flap itself or they can be associated with its donor site. Morbidity from the donor site may be quite significant, such as a ventral hernia following a TRAM flap or a persistent seroma following a latissimus dorsi harvest. Complications involving flaps can be arbitrarily divided into two categories: extrinsic and intrinsic.

Extrinsic Factors

Local Factors

Extrinsic factors are not related to the flap per se and can be a result of either local and/or systemic factors. Local factors are generally any impediment to the circulation of the flap. Classic examples include tight dressings or strangulating stitches that cause circulation impairment. Another example is a hematoma or seroma that compresses the flap pedicle.

CLINICAL CONSIDERATION

Rehabilitation of a patient with a flap begins when the surgeon deems it safe for the patient to perform bed mobility and transfers. Patients with sacral flaps are usually in a fluidized air bed and require longer periods of bed rest than those with extremity flaps. A primary principle of any mobility is to avoid shear, which can disrupt capillary growth and lead to flap failure, undermining, and sinus formation.

Systemic Factors

Systemic factors have an overall effect on the body as well as on the flap itself. Hypovolemia can lead to flap hypoperfusion; hypothermia, as noted earlier, can lead to vasoconstriction. Systemic diseases can also affect flap survival; for example, arteriosclerosis can significantly affect the quality of recipient vessels, therefore decreasing blood supply to a flap especially in the lower extremity. Refer to Chapter 11, Factors That Impede Wound Healing, for other factors that may inhibit wound healing, including the postsurgical healing of a flap.

CLINICAL CONSIDERATION

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Elevation of the extremity with a flap is a basic principle to help avoid venous congestion. This is especially important for any patient with a known history of chronic venous insufficiency. Transfers and positioning are performed with techniques to maintain elevation (eg, the foot is higher than the knee and the knee is higher than the hip) and using equipment that provides support without direct pressure on the flap.

Intrinsic Factors

Intrinsic factors that complicate healing are those related to the flap itself. Aside from an improper flap design or flawed harvest technique, failure is related to poor arterial inflow or venous outflow.

Arterial Insufficiency

Arterial insufficiency refers to a deficit in arterial blood perfusion. This deficit can have its origin anywhere along the vascular pathway of the flap, from the beginning of the pedicle to the end of a perforator. For example, vasoconstriction at the base of the pedicle secondary to sympathetic stimulation can cause arterial insufficiency. However, a flap isolated on a single small perforator can be insufficient to vascularize a large flap. No matter where the occlusion is located, the consequence is the same: inadequate blood supply leads to arterial insufficiency and flap compromise, which is usually followed by partial or complete necrosis. If the arterial insufficiency is partial or limited to a certain area, then partial flap necrosis can occur (**FIGURE 9-19**).

FIGURE 9-19

Example of arterial insufficiency Changes in skin tone to a more pale skin flap and a long capillary refill time (>3 seconds) are indications of arterial insufficiency. The result can be a heavy eschar with necrosis of the subcutaneous tissue. Other clinical symptoms include drainage, loosening of the flap edges, and odor.

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For example, a DIEP flap may have a partial area of fat necrosis. Clinically, a patient may complain of pain and a hardened area in the reconstructed breast.

Venous Insufficiency

Venous insufficiency refers to a lack of venous drainage with blood accumulation in the flap. Similar to arterial insufficiency, the deficit can be anywhere from the capillaries to the pedicle vein(s). Contrary to arterial inflow, venous drainage is often divided into a deep and a superficial system. The proportion of drainage between the two systems is variable among patients and is not always predictable. Since these two systems are not located contiguously to each other, both drainage systems cannot always be harvested at the same time due to flap design constraint. For example, a radial forearm flap can be harvested with two *venae comitantes* (deep venous system) of the radial artery as well as with the cephalic vein (superficial system) without affecting flap design significantly. However, an abdominal flap is harvested with the deep inferior epigastric artery and veins for a DIEP flap or it can be harvested with the superficial inferior epigastric artery and veins for a superficial inferior epigastric artery (SIEA) perforator flap. Because of different anatomic locations, a flap is based on one of the two systems.

The consequence of an inadequate venous drainage is venous congestion (also referred to as secondary or venous ischemia). Clinically, congested flaps appear blue or purple, dusky, and edematous with elevated intravascular pressure in the venous system of the flap. Physically, congested blood occupies volume in the flap, thus increasing intraflap pressure. This elevation can be enough to compress remaining venous outflow (therefore worsening the situation), as well as the arterial inflow, leading ultimately to flap failure. For example, a venous thrombosis has the following sequence of events: (1) venous thrombosis leads to flap congestion, (2) venous congestion leads to compression of arterial inflow, (3) compressed arterial flow causes an arrest in tissue perfusion, and (4) ischemia causes flap failure (**FIGURE 9-20A, B**). Since arterial pressure is significantly higher than venous pressure, blood continues to flow into the flap long after complete venous thrombosis has occurred, resulting in even greater intraflap pressure and

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leading the flap into a *no-reflow* condition with poor outcomes for both the patient and the surgeon. In selected cases, leeches can be applied to the flap to drain congested blood and decrease the overall flap pressure (**FIGURE 9-20C**). However, the appropriate treatment for venous congestion remains exploration in the operating room when possible.

CASE STUDY

FLAPS

A 57-year-old Caucasian woman sustained a necrotizing soft tissue infection of her left lower leg and ankle. One week post-injury, the plastic surgery team was consulted for management of exposed bone, tendons, and joint surfaces. After serial debridement in the operating room, the patient had a 15 ×15 cm soft tissue defect. The patient had no significant medical comorbidities, tissue cultures were negative, and nutritional markers for protein were normal (**FIGURES 9-21** and **9-22**).

DISCUSSION QUESTIONS

1. What is the main goal of the surgery?
2. What kind of coverage is needed for this patient?
3. List specific concerns or important factors in the selection of a durable coverage for this patient.

The key point of this reconstruction is to ensure that all infection has been controlled with surgical debridement and IV antibiotics. The next goal is to provide stable vascularized soft tissue coverage over the exposed bone, tendon, and ankle joint capsule. If it is not reconstructed properly, the patient is at risk for persistent infection and resultant amputation. A skin graft would be insufficient in this case because of the exposed bone and tendon. Wound matrices (dressings) would be inadequate for a defect of this complexity because of the inability of bone and tendon to support granulation tissue needed for re-epithelialization. Therefore, a flap is needed to cover the defect. Because of the location of the injury on the distal third of the leg, a pedicled flap is not an option because it has limited arc of rotation. A free flap is the best option to achieve full coverage of this complex wound.

The choice of flap is surgeon dependent but takes into consideration the size and anatomic location of the defect, thickness of the flap, length and orientation of the pedicle, possibility of subsequent surgeries, long-term aesthetic result, donor site morbidity, and functional aspects of the reconstruction. Of course, patient preference is also important. In this case, a free latissimus dorsi muscle flap covered with an STSG or an anterolateral thigh fasciocutaneous flap would be first-line options. For this patient, a latissimus dorsi muscle free flap was performed.

FIGURE 9-20

Example of flap with venous congestion A. Total failure of a breast flap due to venous outflow obstruction/congestion. **B.** Partial flap necrosis can occur in a flap with venous congestion, as seen on the medial part of the flap. **C.** Venous congestion is sometimes treated with leeches that attach to the flap and suction out the excess venous blood.

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FIGURE 9-21

A lower extremity soft tissue defect post-trauma covered with a latissimus dorsi muscle free flap and a split thickness skin graft.

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FIGURE 9-22

The same flap during the remodeling phase of healing during which the edema noted in the flap can be treated with mild compression to help reshape the ankle and foot.

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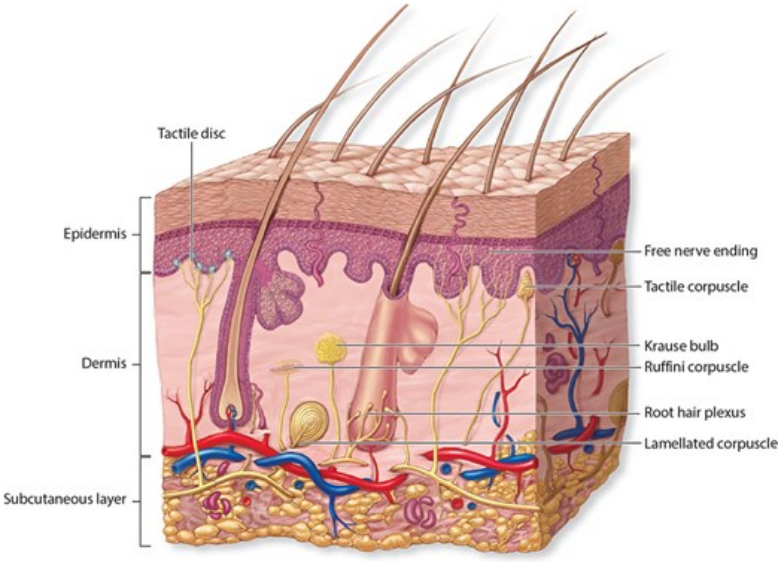
SKIN GRAFTS

Definition

A skin graft, contrary to a flap, does not need a vascular pedicle to ensure its survival. A skin graft is composed of epidermis as well as a variable amount of dermis (**FIGURE 9-23**). Human skin thickness varies greatly within the body, ranging from 0.5 to 4.0 mm.[2] Eyelid skin is among the thinnest of the body; back and scalp skin are the thickest. It is, therefore, the quantity of dermis that is variable in a skin graft. By definition, a graft *is something that is removed from the body, is completely devascularized, and is replaced in another location.*[54] A skin graft can be defined as epidermis and a proportion of dermis completely separated from its donor site that is transferred to a distant recipient bed.

FIGURE 9-23

Skin anatomy Skin grafts can be split-thickness (using the epidermis and part of the dermis) or full-thickness (using all of the epidermis and dermis).



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Classification

By Thickness

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The principal approach to classifying a skin graft is by thickness. A graft that comprises the entire dermis and epidermis is termed a *full-thickness skin graft* (FTSG). If only a fraction of the dermis is harvested with the epidermis, the graft is termed a *split-thickness skin graft* (STSG). Within the STSG spectrum, a graft may be thin, medium, or thick. The instrument used to harvest a skin graft is a called a *dermatome* and can be handheld or power assisted. Mechanical instruments are calibrated to harvest a specific skin thickness, measured in one thousandth of an inch (1/1000 inch) or in millimeters (mm) (**TABLE 9-6**).

TABLE 9-6

STSG Thickness

Thickness	/1000 inch	mm
Thin	8–10	0.020–0.025
Medium	12–14	0.030–0.036
Thick	18–22	0.046–0.056

By Origin

Skin grafts may also be classified by their origin. Grafts can be harvested from humans or animals. If the donor and the recipient of the graft are the same individual, it is termed an *autograft*. If a graft is exchanged between two identical twins, it is termed an *isograft*. When the donor and the recipient are two different individuals but from the same species, it is termed *allograft*. If a graft is exchanged between two different species (eg, pig to human), it is termed *xenograft*.[61]

Autografts are routinely performed as part of plastic surgery procedures. For example, a free latissimus dorsi muscle flap may be covered by an STSG taken from the same patient (**FIGURE 9-24**).

FIGURE 9-24

Free latissimus dorsi muscle flap covered with STSG The split-thickness skin graft on the lower extremity is composed of the epidermis and part of the dermis, thus the red presentation from the presence of skin capillaries. In this patient, both the flap and the skin graft are examples of autografts, meaning the grafted tissue is taken from the patient's own body.

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Use of allografts is more restricted due to the high cost and limited indications. Allografts are harvested from cadavers and distributed for temporary skin coverage (**FIGURE 9-25**). Most allografts are used on burn patients to bridge the period between debridement and final coverage with autografts. If left in place for an extended period of time, a rejection reaction will occur and the skin allograft will be lost.

FIGURE 9-25

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Allografts for temporary wound coverage A. Allograft prior to application. **B.** Allograft applied to an abdominal wound with an enterocutaneous fistula. The goal was to facilitate wound healing prior to resection of the fistula. **C.** Four days after application, part of the allograft is being sloughed off (left edges); in the center, granulation tissue is visible under the allograft that is still adhered to the underlying wound.

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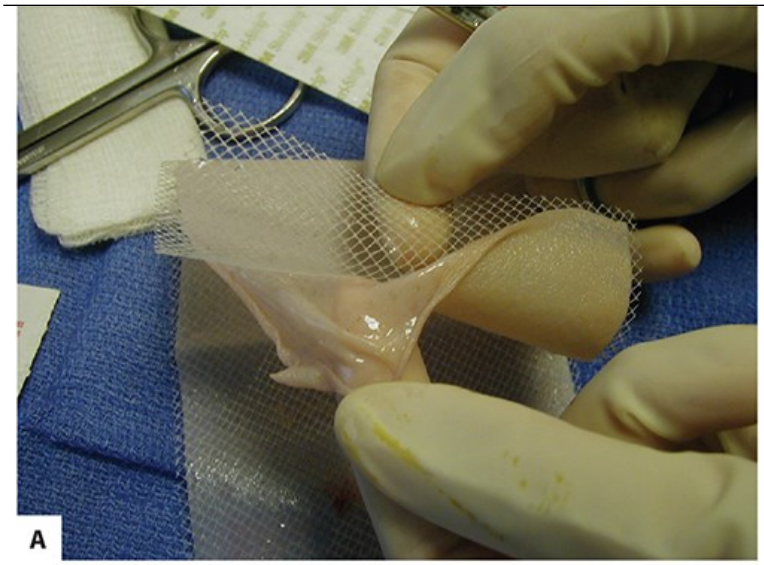

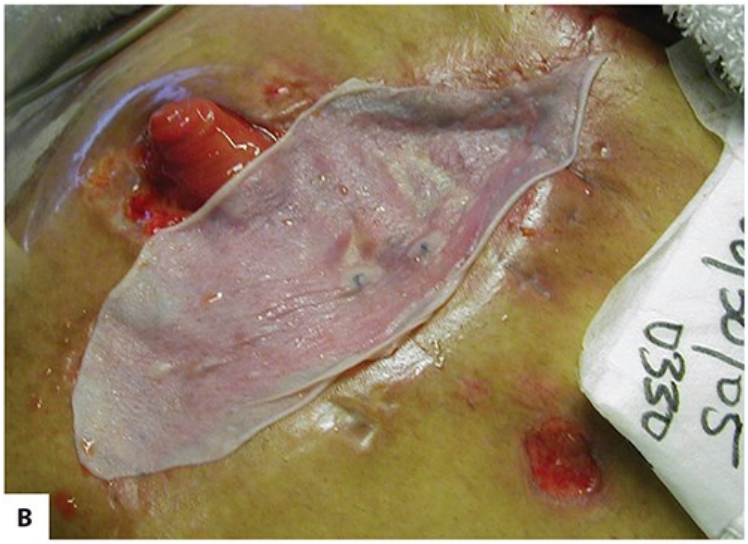

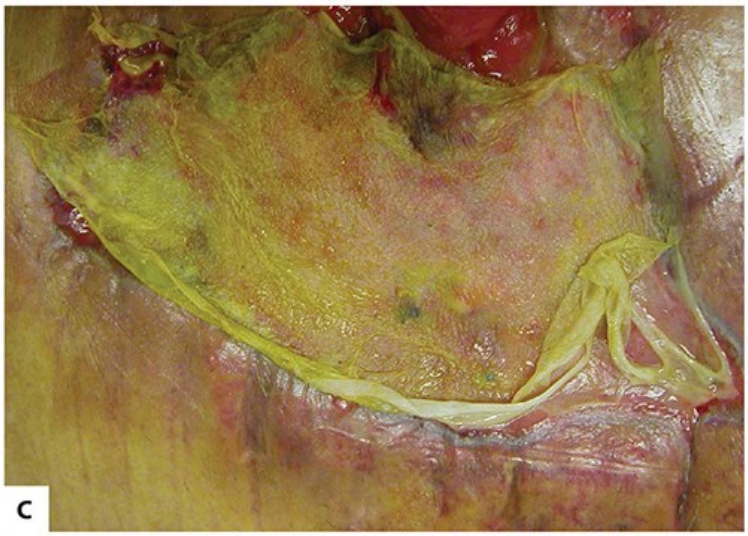

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Because of the morbidity of harvesting skin autograft and its paucity in patients with extensive burns, animals have been considered as a skin source, with the hope of developing a reliable and nonhuman source of skin coverage. Unfortunately, xenografts were found to be expensive and immunogenic; therefore, they may be used as biologic dressings but not for durable long-term skin coverage. Xenograft tissue transplantation is an active area of research. Recently, there has been growing interest in using decellularized animal tissues such as porcine dermis, pericardium, and bladder for wound care indications. However, long-term outcome studies are limited.

Skin Anatomy

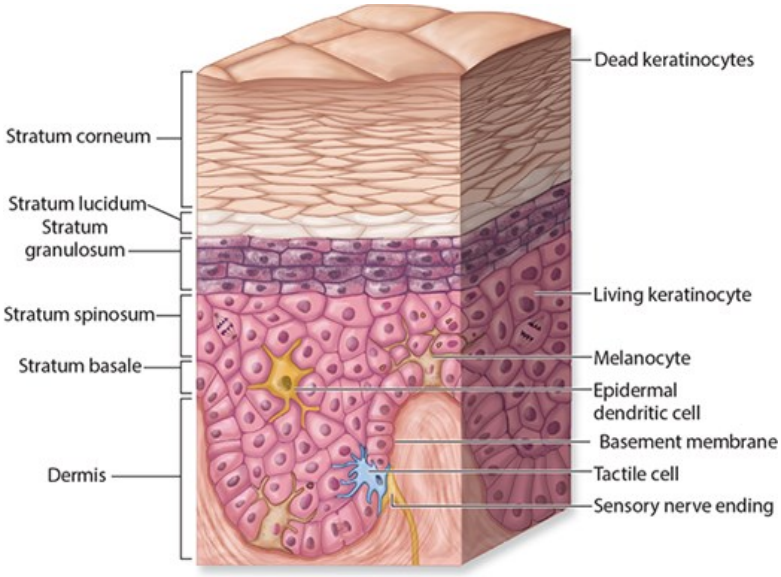
Human skin is the largest organ of the human body and represents 8% of the body, covering 1.2 to 2.2 m^2.[62] Skin is composed of epidermis and dermis, with an underlying subcutaneous fat layer (which is not per se part of the skin). It contains a rich vascular supply, innervation, lymphatic drainage, glands, and hair follicles.

Epidermis and Dermis

The outermost layer of skin is the epidermis and is defined as a keratinized stratified squamous layer that continuously renews itself. It is composed of five different histologic sublayers, with a total thickness of 0.075–0.15 mm (**FIGURE 9-26**).[62] Epidermis is the host of four cell populations including keratinocytes, melanocytes, Langerhans cells, and Merkel cells. Keratinocytes, which are responsible for maintaining a physical barrier against external pathogens, represent at least 80% of epidermal cells.[62]

FIGURE 9-26

Layers of the epidermis.



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The layer between the epidermis and the underlying subcutaneous fat is termed the dermis. It is significantly thicker than the epidermis, ranging from less than 1 mm for the eyelid to over 4 mm for skin on the back.[62] The dermis contains vascular and nervous structures as well as glands and hair follicles (**FIGURE 9-27**). Fibroblasts are the principal cell population of the dermis, along with macrophages and adipocytes.

FIGURE 9-27

Vascular anatomy of dermis The dermis contains networks of capillaries, termed plexuses. The upper horizontal network is in the papillary dermis, and the lower horizontal plexus is at the dermis–subcutaneous tissue junction. Random skin flaps that are not supplied by named axial blood vessels are viable secondary to perfusion from both dermal plexuses.

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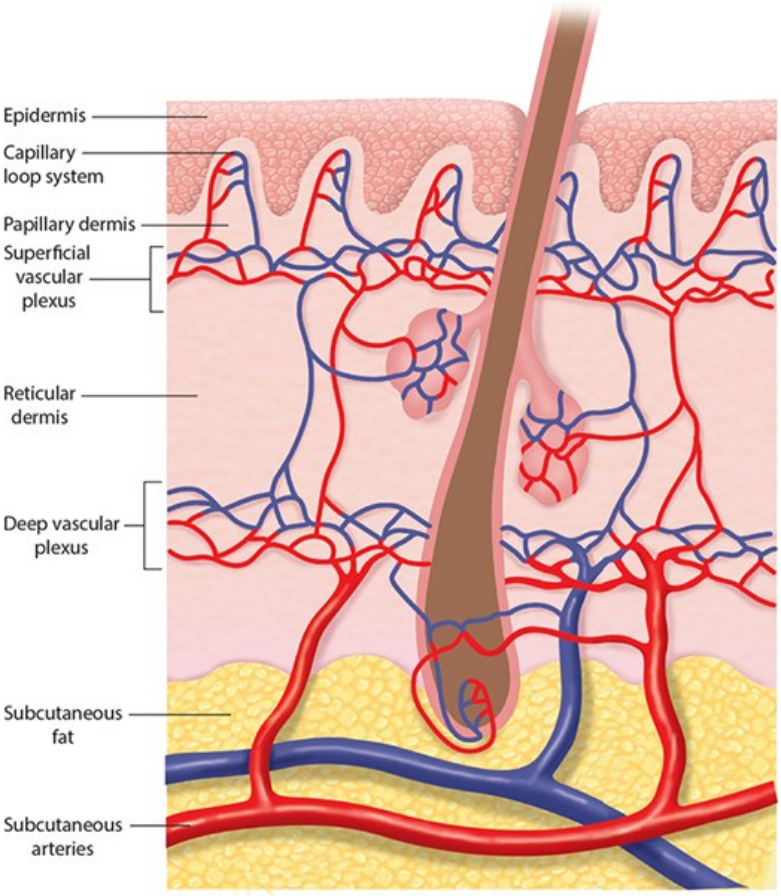

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Dermal cells are contained in the extracellular matrix (ECM), which acts as a support for cells within the dermis. ECM is composed of collagen and gel-like substances, including glycosaminoglycans, proteoglycans, and glycoproteins. The non-collagenous component of ECM is referred to as the ground substance. Fibroblasts are responsible for production of collagen fibrils as well as components of the ground substance. Approximately 75% of the dry weight of skin is collagen. There are many types of collagen but type I is the most abundant in the dermis.[62] Collagen is responsible for the tensile strength and elasticity of the skin, along with elastin which is another protein synthetized by fibroblasts. Elastin allows the skin to stretch and return to its resting position without deformation. The dermis is divided into a superficial layer termed papillary dermis and a deeper layer termed reticular dermis.

Blood Supply

As illustrated in the first section of this chapter, skin is vascularized by networks of blood vessels termed plexuses. The arterioles and venules within the dermis form two horizontal plexuses: an upper horizontal network in the papillary dermis and a lower horizontal plexus at the dermis–subcutaneous tissue junction.

Skin Appendages

The various glands and hair follicles located in the dermis are termed *skin appendages*. The pilosebaceous units, comprising hair follicles and sebaceous glands, are located in the vast majority of the skin, with the exception of mucous membranes, palms, and soles. Eccrine sweat glands are ubiquitous on skin except for mucous membranes. Apocrine sweat glands are localized in specific locations, namely the axilla, perineum, areolae, eyelid, and external auditory canal.[62]

The relevance of skin anatomy in skin graft harvesting is important; for example, a STSG of 12/1000 inch (0.03 mm) from the back includes epidermis and part of the papillary dermis; however, hair follicles and sweat glands are not harvested with the split-thickness skin graft. The consequence is that

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the grafted site will not have hair nor glandular structure, and the donor site, after healing, will regrow hair and keep its glandular potential. An FTSG from the abdomen that is transferred to the axilla carries all of the skin appendages to the newly grafted site. Thus, if hair were present on the abdomen, then hair would grow in the axilla, assuming an uneventful healing of the skin graft. Also, after STSG harvest, numerous small foci of bleeding are present representing the tangential cut of the upper horizontal plexus.

Physiology

Understanding the skin graft survival physiology is of paramount importance since it allows the surgeon to carefully prepare the recipient site of the graft and adequately immobilize the graft after placement. The first step before harvesting any skin graft is to prepare the recipient site. Since a skin graft does not have any intrinsic vascular supply when transferred, it relies solely on the resource of the recipient bed for survival during the postoperative period. An adequate recipient site must, therefore, be well vascularized, devoid of necrotic material, and free from pathogenic agents. Failure to achieve a proper recipient site will result in "non-take" of the skin graft, resulting in failure for the patient and the surgeon (**FIGURE 9-28**). Following are examples of patients who would not be good candidates due to high risk of failure:

- *Patient with a lower extremity trauma with exposed tibia stripped of its periosteum.* The periosteum is essential to deliver nutrients to the graft and bone devoid of periosteum cannot be an adequate recipient bed.
- *Patient with burn wounds that are colonized with Staphylococcus aureus.* The wound may appear ready for skin graft after debridement; however, chances of failure are high because the graft may be inhibited by bacterial-derived collagenases.
- *Patient who is taking high doses of steroids or anti-inflammatory medications.* The same medications that inhibit wound healing will cause a graft to have a high risk of failure.
- *STSG on a plantar ulcer for a patient with diabetes and severe peripheral vascular disease.* The graft will not receive enough nutrients to survive and may be subjected to both friction and shear forces if the graft is not adequately off-loaded.

FIGURE 9-28

Skin graft with areas of both normal "take" and "non-take."

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The normal healing of a skin graft goes through three steps: serum imbibition, inosculation, and revascularization.

Serum Imbibition

At the time it is harvested, a skin graft loses its vascular supply. In order to survive, a skin graft needs to receive nutrients from the recipient bed. Vascular connections cannot be re-established instantaneously; therefore, during the first 48 hours oxygen and nutrients diffuse passively from the recipient bed to the skin, a period termed the *serum imbibition* phase. Fibrin fixes the skin graft to the wound bed as fibrinogen is transformed into

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fibrin. During this phase, skin grafts gain up to 40% in weight compared with their initial weight in the first 24 hours; however, this gain is only 5% 1-week postgrafting.[63,64] Thickness of the graft is important, as thicker grafts need more diffusion of nutrients to survive. A poor recipient bed may have difficulty feeding a skin graft, for example, the plantar ulcer in a patient with vascular disease.

Accumulation of fluid in the interface between the bed and the graft, termed a *seroma*, affects survival of the graft because nutrients have to pass through the seroma to reach the graft. Clinically, a localized area with a seroma within a skin graft translates into a graft failure in the localized area. In addition, during this phase skin grafts need to be protected from external trauma because any shearing of the graft may compromise its outcome.

Inosculation

The second phase of skin graft healing takes place between 48 hours and 5 days post-surgery and is termed *inosculation*. Inosculation originates from Latin, *in* and *osculare*; it can be translated as "to unite by a small opening." It was originally thought that capillaries in the basal membrane would realign and connect with the skin graft; however, the process is in fact quite complex, involving anastomosis, neovascularization, and endothelial cell proliferation.[64] This phase marks the beginning of the process to achieve "normal" circulation by creating the foundation for a more definitive vascular support of the graft. Clinically, the graft appears congested and swollen. During this phase, the graft is still fragile and must be protected by a dressing. Any trauma, especially shearing of the graft, would disrupt the tiny capillary network developing to revascularize the graft.

Revascularization

The final phase of skin graft healing is revascularization during which vascular channels form afferent and efferent vessels, as well as lymphatic drainage. The swollen appearance disappears and is replaced by a more natural texture and color. This is also a maturation phase during which contraction and remodeling of the surrounding tissues take place. Skin grafts take at least 1 year to complete maturation, and can take even longer in burned victims and children.[65] Long-term contraction of the graft varies according to its thickness. Skin graft contraction is divided into two types: the immediate contraction after harvest (termed *primary contraction*) and the long-term contraction after maturation (termed *secondary contraction*). Immediately after harvest, the thinner the graft, the lesser the amount of contraction; a full-thickness skin graft (FTSG) contracts the most. However, during the maturation phase, thinner grafts contract the most while FTSGs contract the least (**FIGURE 9-29**).

FIGURE 9-29

Healed STSG and FTSG The plantar surface of the foot illustrates both a split-thickness and a full-thickness skin graft in the maturation or remodeling phase of wound healing.

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Graft Immobilization

As mentioned earlier, skin grafts need to be protected from external distress to increase graft survival. Since contact of the graft with the wound bed is

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important, especially for the serum imbibition phase, a typical skin graft dressing includes a degree of pressure on the graft. If the wound bed is not a flat surface and is irregular, the graft has to be secured to the wound bed with stitches or staples in order to maintain close contact and prevent seroma or hematoma accumulation. Lack of appropriate contact between the graft and the recipient bed predictably results in graft failure.

Bolster Dressing

The classic skin graft dressing, termed a bolster dressing, is simply a method to maintain close contact of the graft to the recipient bed and to protect it against trauma, primarily shearing forces (**FIGURE 9-30**). An example of a bolster dressing consists of a nonadherent dressing on the skin graft such as an Adaptic (Johnson & Johnson, New Brunswick, NJ). This layer maintains a moist environment and prevents tearing or ripping of the graft during dressing removal. The dressing is then padded by a cotton ball layer to perfectly "mold" to the contour of the skin graft. The last step is to use sutures to ensure no movement of the dressing.

FIGURE 9-30

Example of a bolster dressing A bolster dressing is used to maintain good contact of the graft with the recipient wound bed and to protect it against trauma. In this case, the graft is covered with several layers of nonadherent Xeroform gauze and sutured in place to ensure no movement of the dressing.

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Negative Pressure Therapy

Negative pressure therapy dressings are also used to protect skin grafts. This dressing maintains adequate graft-bed contact, protects the graft from trauma, and removes excess fluid by the negative pressure therapy. A negative pressure of 75 mmHg is normally sufficient to allow graft survival. Unfortunately, this dressing is more costly, involves carrying the pumping device, and cannot be used in all areas of the body. For example, a cheek skin graft would be most likely dressed with a bolster dressing rather than a negative pressure therapy device, as the seal needed for the vacuum to be created is hard to achieve in this area. (Refer to Chapter 15 for more information on negative pressure wound therapy.)

Skin graft survival, however, can be achieved without any dressing depending upon the location and functional activities of the involved body part. For example, an STSG on a latissimus dorsi free flap typically is not covered by bolster or negative pressure therapy dressing; pressure on the graft may compromise flap survival (**FIGURE 9-31**). As long as the skin graft is protected from trauma, is kept moist, and has no seroma or hematoma accumulation, it should survive if the recipient bed is appropriate. In the free latissimus dorsi example, grafts are often expanded (see the section "Graft Expansion") to prevent fluid accumulation and minimize the amount of skin needed. Muscle is an excellent recipient bed; in fact STSG on a well-vascularized muscle adheres in only a few minutes due to fibrin exudation. In addition, fibrin glue may be used to allow a better contact between the graft and the recipient bed and is sprayed on the wound bed before apposing the skin graft.

FIGURE 9-31

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STSG on a free latissimus dorsi muscle flap A. Basal cell carcinoma of the scalp. **B.** Postoperative result after wide excision of tumor and reconstruction with free latissimus dorsi flap and STSG.

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The length of time required for a skin graft postoperative dressing arises from an understanding of graft healing. The first 5 days are critical for graft survival as discussed earlier; thus, dressings are normally removed after 5 days. The graft still needs to be protected after initial dressing removal because it may be damaged if subjected to trauma, especially shear or friction forces. The maturation process is variable among individuals and clinical evaluation is warranted to assess adequate healing. Of note, some clinicians do not wait 5 days; they prefer to remove dressings earlier and evaluate the graft for fluid accumulation, position, or any other problem. If accumulated fluid is drained or graft repositioned early, graft survival can still be achieved.

Graft Failure

Unfortunately, skin graft survival may be partial, leaving an area where the graft does not take to its underlying bed. These areas can be regrafted or left to heal by secondary intention. Skin grafting demands a meticulous harvesting, insetting, and dressing technique. Any failure along the chain of events from harvesting to dressing will lead to skin graft failure (**FIGURE 9-32**).

FIGURE 9-32

Examples of skin graft failure Impending skin graft failure may be detected by changes in skin color, eg, dark red indicates hematoma formation beneath the skin; white, infection; black, necrosis; yellow slough, autolysis of nonviable tissue. **A.** The dark edges, loss of graft at the left edge, and dusky color of the skin around the lower incision are signs that part of the graft did not take. **B.** The white nonviable tissue in the graft site indicates possible infection. **C.** Remnants of meshed graft, along with large amounts of slough and necrotic tissue, are observed in this dehisced surgical incision after a hernia repair.

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Factors of Skin Failure

Skin graft failures have multiple origins, and the first cause is a technical error, eg, harvesting the skin graft too thick; placing the epidermal side of the skin to the recipient bed (rather than the dermal side); not placing the graft properly in the recipient bed, thus allowing movement or not creating close contact with the bed; and applying a dressing inadequately so that the graft is not properly protected from friction or shear. Again, a meticulous technique is mandatory to achieve skin graft survival.

A second cause of skin graft failure relates to fluid accumulation between the recipient bed and the skin graft. Fluid accumulating in the interface disrupts the serum imbibition process and causes skin graft failure. Seromas (clear fluid) or hematomas (blood) originate from the recipient bed; it is therefore essential to have a properly prepared bed. Adequate hemostasis prevents hematoma; drainage holes in the skin graft prevent seromas.

Drainage holes in a skin graft are performed in various ways. Skin expansion (see the next section), with its meshed network, allows easy drainage of any fluid. But drainage pits can also be performed by creating holes or lacerations in the skin graft. This process is termed *pie crusting* and is performed using a scalpel or fine scissors. The number of holes or incisions is dictated by the size and localization of the skin graft. Ultimately, openings in the skin graft will heal by secondary intention, causing small areas of scarring in each of the holes or lacerations. However, for cosmetic reasons a skin graft can be applied without any drainage openings. For example, an FTSG on the face generally does not have pie crusting; attention must then be focused on a recipient bed with proper hemostasis and a good bolster dressing with close contact between the graft and the bed.

Infection, either bacterial or fungal, is another potential cause of skin graft failure. Typically, a count of more than 10^5 organisms per gram of tissue leads to graft failure. The exception is *Streptococcus* infection, where less than 10^5 organisms per gram of tissue are needed to cause graft failure. When infection occurs, purulence may accumulate between the graft and the bed, thereby preventing revascularization. When *Streptococcus* is present, enzymes secreted by the microbes disrupt the fibrin layer between the graft and the bed. Skin grafting without proper antimicrobial treatment will lead to skin failure.

Finally, a poor recipient bed with inadequate vascular supply that is insufficient to nourish the graft will result in failure. Venous congestion, arterial insufficiency, and lymphedema are examples of factors that may be responsible for a poor recipient bed.

Skin graft failure can be observed months after grafting. Contraction of the skin graft can be very important and may turn what appears to be a healthy graft into a functional failure. Burn units, with especially challenging patients, can observe this phenomenon. For example, an axillary region can be grafted with an STSG only to realize, after months of healing, that arm movement restrictions are present due to skin contraction.

Graft Expansion

Skin is a scarce resource, especially for severely burned patients. For cosmetic reasons, there are several techniques that can be used to conserve skin and minimize scarring of the harvest site. An elegant way to expand the surface of skin grafts is to create multiple windows and stretch the graft to increase the total surface area. This process is termed *graft expansion* and is accomplished with a *skin graft mesher* that uses blades with built-in cutting ratios. For example, a blade with a skin ratio of 1.5:1 expands harvested skin 1.5 times the initial surface area. Blades with ratios up to 9:1 are available. A blade with a higher ratio allows for more expansion but also leaves larger open areas in the graft.

Healing of the meshed skin graft occurs in two different ways. The expanded skin heals as previously described with open areas healing by second intention. Keratinocytes from the margin of the expanded skin colonize and fill open areas. Aesthetically, a meshed skin graft may have an unequal and rough appearance with multiple asperities (**FIGURE 9-33**). If hypergranulation is present, the result can be quite displeasing. The anatomic location of the skin graft recipient site often guides the type of meshing. For example, a face skin graft will not be meshed while a skin graft over a muscle-free flap for a lower extremity trauma may be meshed (**FIGURE 9-34**).

FIGURE 9-33

A meshed skin graft A meshed skin graft immediately after placement over a lower extremity traumatic wound. The meshed texture of the skin graft allows the harvested skin to be stretched to cover a larger area than the donor site, thereby minimizing the size of the donor wound.

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FIGURE 9-34

Expanded mesh skin graft on the lower extremity A meshed graft that allows drainage to escape is especially important on the lower extremity where the wound may interfere with lymphatic flow and result in edema below the graft site. Modified compression with short stretch bandages may help manage edema, improve the graft take, and result in a better outcome.

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As mentioned earlier, the meshed area permits evacuation of fluid that may cause loss of contact between the graft and the recipient bed. A compromise between a better aesthetic result and a functioning drainage system is to mesh the graft without expanding it. Since the open areas heal by second intention and create an unpleasing appearance, an unexpanded mesh graft prevents fluid accumulation while at the same time minimizing scarring.

Donor Site Selection and Morbidity

An autologous skin graft is usually harvested from a body site that is distant from the recipient defect. The consequence is termed *donor site morbidity*. Selecting an appropriate donor site is not trivial and demands careful consideration. Ideally, a skin graft would exactly match the skin it replaces; it would have the same color, texture, thickness, and appendages. Unfortunately, this is rarely achieved.

The main consideration in donor site selection is the area that it will cover. Certain areas dictate an FTSG while others can be accommodated with an STSG. The face generally needs an FTSG for aesthetic concerns. In this example, color is a major factor in selecting a donor site. A distinctly different color in an area of the face attracts the eye and is cosmetically displeasing. Skin from the pre- or postauricular region or the supraclavicular region is often selected as a donor site for a facial skin graft.

However, a skin graft to cover a lower extremity trauma may not carry as many aesthetic considerations; also the same leg is often selected as a donor site. For example, a skin graft needed to cover a calf defect may be taken from the thigh region of the same leg.

FTSG donor sites are usually closed primarily. Since all of the dermis is taken with the graft, leaving the donor site open would lead to secondary intention healing, thus creating a scar. For example, an FTSG can be harvested from the groin region to fill an axilla skin defect. The skin deficit in the groin region is then undermined to allow primary closure. For an STSG, part of the dermis is left on the donor site and is key to healing, which occurs in

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7–21 days[66] by a process called *re-epithelialization*. The source of precursor cells comes from the periphery of the donor site and from the hair follicle stem cells. Once healed, an STSG generally leaves a distinctive texture that is different from the surrounding skin so that it is noticeable; it tends to have a shiny or polished appearance and is less pliable. Pigmentation is often affected, ranging from hypo- to hyperpigmented (**FIGURE 9-35**). If appendages have been incorporated into the graft, they are not replaced. Ultimately, the overall thickness of the graft compared to the total dermis thickness is a significant factor in the final look and feel of the donor site.

FIGURE 9-35

Hypopigmented or reddish healed donor site Two donor sites on the thigh illustrate the color changes that occur. On the left, the hypopigmented area is from an older donor site that is completely healed, and on the right, the signs of neoangiogenesis give the donor site a reddish vascular appearance. As the re-epithelialized tissue remodels, the red scar tissue will diminish and the site will probably become hypopigmented.

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Unfortunately, donor site healing can be affected by common complications affecting any type of surgery (**FIGURE 9-36**). For example, a donor site can bleed excessively, be infected, or develop a hypertrophic scar/keloid. Meticulous wound care with occlusive dressings, with or without antimicrobial agents, are helpful in preventing donor site complications and in minimizing pain with dressing changes and functional activities. An informed discussion with patients about all of the aforementioned potential complications is essential to achieve a successful result.

CASE STUDY

STSG

A healthy 30-year-old African American male sustained burn injuries to his hands and forearms while trying to start a campfire with gasoline. Because of the location of his injuries, he was transferred to a burn center. Evaluation of his injuries revealed a combination of superficial and deep partial-thickness burns. Initial treatment was debridement and moist dressings. He had no other injuries.

Superficial burns heal by re-epithelialization leaving no scar, but deep partial thickness burns heal by second intention, creating scars if not treated.

DISCUSSION QUESTIONS

1. What is the most important treatment for a burn injury?
2. Why is skin grafting important in the overall medical management of this patient?
3. List pros and cons of an STSG versus FTSG treatment for this patient.

The key point of the reconstruction for this patient is to remove all nonviable tissue (debridement) and prevent limiting and disfiguring scarring. The best way to achieve this result is by skin grafting the lesion, and STSG is preferred over FTSG. STSG can be taken from any normal skin area of the body and the donor site will re-epithelialize. Thin skin grafts have a better “take” than thicker skin grafts due to the initial diffusion process involved in the first days post-grafting. FTSG donor sites have to be closed primarily, thus limiting the overall surface area that can be collected. FTSG is not an option

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for this patient.

The location from which the STSG is taken is determined after consulting the patient. For a patient with dark skin, hypo- or hyperpigmentation post-STSG is possible and has to be discussed with the patient. In this case, STSG would be harvested from the thigh.

FIGURE 9-36

Donor site complications The most likely complication of donor sites is infection, as observed in **A** in which a large amount of slough and drainage are preventing re-epithelialization. The lateral thigh donor sites in **B** and **C** had two less frequent complications, an allergic reaction to silver dressings used to address what appeared to be critical colonization and friction from the arm rest of the wheelchair.

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The ultimate outcome for any skin graft is to have an aesthetically acceptable appearance and to recover full function of the affected part (**FIGURE 9-37**).

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FIGURE 9-37

Long-term outcomes for an STSG The goal of every STSG is to have an aesthetically acceptable outcome for the patient. The skin graft on this patient's scalp is barely visible; however, the full healing process from onset through maturation can take up to 2 years.

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SUMMARY

Free flaps are defined as units of tissue that may be transferred from donor to recipient sites while maintaining their blood supply. Flaps can be defined by their vascular supply, tissue composition, methods of transfer, or vascular orientation. Free flaps can be defined by the relevant vascular anatomy, including muscle flaps, fasciocutaneous flaps, and perforator flaps. The concept of angiosomes and venosomes further defines the vascular anatomy and circulation required for flap survival. Basic physiology concepts specific to the intrinsic nature of free tissue transfers include local and systemic effects after a free flap harvest. Flap monitoring techniques, including physiological parameters and ancillary methods (Doppler, oximetry, and tissue staining), are used to monitor flaps after surgery. Extrinsic and intrinsic factors can lead to flap failure.

Skin grafts are classified as full or partial thickness. The normal process of skin graft healing includes serum imbibition, inosculation, and revascularization. Factors affecting graft survival include proper immobilization after surgery, meticulous technique, prevention of infection, and adequate vascularization.

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